BRAIN.

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Original Articles and Clinical Cases.

STUDIES ON THE LESIONS PRODUCED BY THE ACTION OF CERTAIN POISONS ON THE CORTICAL NERVE CELL.—I. ALCOHOL.

BY HENRY J. BERKLEY, M.D. (BALTIMORE, U.S.A.)

From the Anatomical Laboratory of the Johns Hopkins University.

PART I.

Section I.—Experimental Lesions produced by the Action of Absolute Ethyl Alcohol on the Nerve Cells of the Rabbit's Brain.

THE most extensive treatise on the pathology of chronic alcoholism to be found in the English language, is Bevan Lewis' article in his text-book on "Mental Diseases." Indeed, so widely has it been accepted as the minute anatomy of this morbid state, that in Tuke's "Dictionary of Psychological Medicine," it is quoted to the exclusion of all other writings. Abstracted, it reads as follows:—"The changes in the vessels are nuclear proliferation, atheroma, and aneurysmal dilatation, the last eventually giving rise to a cribriform condition. The motor cells are swollen and rounded, stain deeply, become granularly pigmented, and the apical processes degenerate. The cellular wall is thickened. There is a considerable quantity of pigment deposited between the shrinking protoplasm and this cell wall. The processes of the cells are stunted, and are covered with nuclei, and the

protoplasm is granular or vacuolated. In the lowest layer the scavenger cells and nuclei cover the spindle nerve cells, which are very much degenerated and altered, and are practically being devoured by these proliferating cells.

"The medullary sheath of the nerve processes gradually disappears, or is so altered by the invading connective tissue that the axis-cylinder—which is frequently fusiform, as in other cases of inflammation of the nerve fibre—can be perfectly well stained with the aniline colour, when it becomes a prominent feature in the cortex."

Lewis describes the motor cells of the so-called third layer as being the first implicated in the cortex, and that those of the deeper and more superficial layers follow in order.

It would seem from this description as if there were no specific lesions of the central nervous system, from the continued abuse of alcohol, to be discovered; certainly, the vascular changes found are frequent in brains that have exhibited no clinical picture of the effects of alcohol during life, and taking atheroma and miliary aneurisms from among the list, it would appear as if there were other conditions of which we at present know nothing; as all pathologists are familiar with brains that show in marked degree these changes, but whose owners have in no wise been addicted to the abuse of the drug. Nevertheless, there can be no doubt that alcohol, in the common forms of wines and distilled liquors, does exert a direct influence on the cerebral vessels, irritating their coats, especially the intima: perhaps paralysing the vaso-motor nerves accompanying them in more or less enduring fashion, hence giving rise, from the irritation, to nuclear proliferation, and from the distension of the vessels, to transudations, local minute extravasations, clumps of hæmatoidin granules, and detritus in the peri-vascular sheaths; then blocking of the lymph currents, and general disturbance of the lymph flow in the peri-vascular and peri-cellular channels.

Of the changes in the nerve cell produced by the direct contact of the diluted drug flowing with the nutrient serum from the blood channels into the peri-cellular spaces, we at present know nothing; but from a clinical standpoint, the

immediate effect in occasioning disturbed metabolism must be very great.

With Lewis' ideas of the coarser changes produced by the continued abuse of the drug upon the nerve cell, we are in unison, as only too frequently in alcoholic brains have we met with the shrunken protoplasm, stunted processes, and granular cell bodies; but these alterations mark only the after-effects of long continued malnutrition and irritation of the cells by the poison. In other words, we see only the later effects of a degeneration which has preceded a considerable distance toward utter deterioration of the cell body before it is visible to carmine, hæmatoxylin, aniline blue-black, and the freezing methods. What we really wish to ascertain is not only the condition of the protoplasmic body, but also the early changes in the finest twigs of the dendrites, and the finest variations of the nucleus and nucleolus from the normal.

A newer though very small literature has sprung up since 1890, when the work of Lewis first appeared, and the methods of histological research have been extended to a realm whose boundaries were only beginning to be entered upon at that date. I refer to the beginning of the extended use of the chromate of silver methods, and the magenta and methylene blue stains of Nissl—the one almost fully supplementing the other, in so far that by one we may obtain perfect impregnations of the neuron to the uttermost limits of the dendrites, and by the other an accurate view of the condition of the cellular protoplasm, and in particular the nucleus with its chromophile particles.

The newer literature, as already mentioned, is very scant; portions of three articles only treat of the lesions of the nerve cell when subjected to the presence of alcohol in the living organism.

Colella (*Arch. Ital. de Biologie*, 1894, p. 216), by the application of the Golgi silver method to the cortex of an alcoholic brain, found lesions of the neuraxon of the nerve cell, slight involvement of the cellular body, and dendrites in the alteration. He speaks of the blood vessels and neuroglia as not being implicated in the changes.

Vas (*Arch. f. Exper. Path. u. Pharmakologie*, Bd. xxxiii., S. 141), in an experimental work on the poisonous effects of alcohol and nicotin on the nerve cells, finds well-marked changes in these bodies, especially in the great cells of the anterior horns of the spinal cord, and in the spinal and sympathetic ganglia. As a first change, the cells lose their chromatin structure; the finely granular character is perceptibly diminished, and gives place to a homogeneous swelling. In advanced cases of the alteration, the cellular protoplasm alters its power of taking up the colouring matter, and appears darker than normal. A shrinkage of the protoplasmic body cannot be definitely ascertained.

The study of Andriezen (BRAIN, 1894) is of greater interest than the foregoing works, and is worthy of careful consideration. Using a modification of the Golgi method, he finds extensive changes in the neurodendrites and neuraxons of the nerve cells of the cortex, the alteration beginning in the fine bead-like "contact granules" upon the substance of the protoplasmic extensions, proceed to moniliform swellings along the course of the protoplasmic twigs. With the further progress of the lesion, these softened and enlarged protoplasmic masses form irregular botryoidal masses, mainly clothing the now irregularly bared protoplasmic stem, which rapidly becomes attenuated.

"The changes in the cell body are of similar nature, and consist in a gradual disintegration after the apical and others of the processes have suffered. Here and there at the side of the cell body the protoplasm seems to become frayed, or as it were eroded. In other cases the cell protoplasm becomes vacuolated from within, and the process continues until the whole interior protoplasmic structure is channeled by holes and seams of liquefaction."

The nerve fibres of the cortex Andriezen finds granular and wrinkled in outline, and what is much less certain, a process of unequal staining, portions of the filament being completely stained, while others are pale or colourless, and only visible by the highest powers of the microscope.

It will be noticed that in none of these articles is there any mention of the state of the nucleus and nucleolus. This

partly arises from the methods of staining used in the various investigations. Lewis' aniline blue-black does not allow the interior structure of the cell to be accurately seen, nor does the silver method permit any inquiry in this direction, and Vas, who used nuclear stains, does not seem to have considered the nucleus of the nerve cell, probably from the difficulties that attend its accurate investigation. The material at our command has, however, allowed of more accurate procedure, and in this direction our labours have been partly directed, somewhat to the exclusion of the intimate structure of the nerve cells protoplasm.

Thanks to the kindness of Prof. Welch and Dr. Friedenwald in furnishing me with the brains of a number of rabbits that had been subjected to the continued—*intra vitam*—action of absolute ethyl alcohol over a considerable space of time, our material allowed us a better opportunity for the investigation of the nerve cells than could be obtained from human alcoholic brains, in so far that we had an accurate knowledge of the amount of alcohol the animal had been subjected to per kilo of bodily weight, the duration of the poisoning, and last, though not least, we were enabled to obtain the cerebra in much fresher condition than could be obtained from the autopsy table. Appended is a condensed table of the histories of the five rabbits used in the investigation.

CONDENSED RABBIT HISTORIES.

No. of Rabbit.	Apparent Age.	Weight in grammes.	Quantity of Alcohol fed per diem.	Began to feed.	Died.	Weight at time of death.	Clinical cause of death.	Gross anatomical lesions found.
6	Adult	1450	5 to 8 cc.	Dec. 19, 1893	Dec. 20, 1894	1420	Convulsions	Fatty heart
10	,,	1920	,, ,,	Dec. 30, 1893	Oct. 27, 1894	2500	Ascites	Cirrhosis of liver
39	,,	1540	,, ,,	July 5, 1894	Dec. 5, 1894	1400	Convulsions	Fatty heart
41	,,	1890	,, ,,	July 5, 1894	Feb. 4, 1895	1420	,,	,, ,,
42	,,	1940	,, ,,	July 5, 1894	Feb. 10, 1895	1640	,,	,, ,,

Methods of Examination.

All the specimens of brain tissues that came into my hands for the purposes of this study—pathological as well as normal—were hardened either in alcohol or in Müller's fluid. For the Nissl method and modifications thereof used for the demonstration of the cellular structures, and for hæmatoxylin-eosin staining for the blood vessels, the former method of hardening was applicable, but for studying the finer dendrites of the nerve cells it became necessary to devise some ready method of staining by which impregnations with metallic salts could be obtained for tingeing the finer extensions of the nerve cells. A research among the literature devoted to microscopic technique failed to afford any suitable process, and after consideration, the following process was adopted and found to be more satisfactory than any other, and, indeed, it gave finer and more complete pictures of the cortical neurons than the ordinary Golgi method. We have at this time stained a large number of pieces of brain substance with it, both human and rabbit, and have yet to record a single failure in the process; though, of course, all the impregnations were not equally good. The sole disadvantage the method has against the Golgi-Cajal procedure is, that the thicker dendrons are rendered so brittle by the silver that they are readily broken into fragments by the sharp edge of the knife passing through the tissue, but as the fragments are not usually carried far from their natural seat, errors from this source are to be avoided with ease.[1] On the other hand, the great advantage that accrues to the pathologist from being able to make use of any desired portion of an entire brain already hardened after the usual manner in Müller's fluid, is at once apparent, particularly when the silver impregnations obtained are much finer than those had after a troublesome and particular hardening process, and though it is but a small consideration, it is more economical of osmic acid than the Cajal method, as the osmic acid mixture may be used more than once.

[1] We found in later preparations that the cause of this brittleness was overhardening in the Müller's fluid, and could easily be avoided.

The staining obtained is much more intense than is found after the ordinary chrome-silver staining, the cell bodies and processes are intensely black, and it is only at the points of the finest dendrites that the familiar reddish-black colour makes its appearance. It is evident from the appearance of the nerve cells and axons that the chemical combination between the chrome saturated nerve tissues, and the silver salts is quite different from the simple chrome-silver combination of the Cajal process. Our method is a much closer approach to a true stain than an impregnation, and no traces of an incrustation are ever to be found. Under intense illumination the cell body is found to be homogeneously blackened, and fine lines are seen running through the protoplasm from one pole of the cell to the other. The addition of sodium sulphate and phospho-molybdic acid to the hardening and staining fluids respectively, makes the chemical reaction between the constituent elements much more complicated than when there are only the simple kalium bichromate, osmic acid, and silver nitrate to deal with, and an attempt to formulate the resulting reactions would be most difficult.

The process is as follows :—The cerebra are treated with Müller's fluid until the tissue is of sufficient consistency to admit of fairly thin sections. The portions of the brain selected are then cut into pieces not more than three millimetres in thickness, and the slices are immersed in a mixture of a three per cent. solution of bichromate of potassium, and solution of one per cent. osmic acid, in the proportion of one hundred parts of the former to twenty parts of the latter. In this mixture the slices lie from three to five days, are then removed from the fluid, and slightly dried on filter paper to remove any superfluous bichromate, are washed for a few minutes in a weak solution of silver nitrate, and then go into the second or staining mixture, which is made by adding two drops of a ten per cent. solution of phosphomolybdic acid to each sixty cc. of one per cent. arg. nitrate solution in distilled water.

The second solution is to be made only as needed, and at the moment before placing the brain tissue in it. In the

mixture of finely precipitated phosphomolybdate of silver held in suspension in the free nitrate of silver solution, the tissues are immersed, and allowed to remain without being disturbed for two or three days, or if it is inconvenient to cut them at this time, a few drops of fresh silver solution may be added to the old fluid, and in this the tissues may remain indefinitely. The object in adding the additional silver nitrate is to prevent any chance of all the salt being precipitated or absorbed, as plain water will soon cause a deterioration of the distinctness of the impregnation of the nerve cells. Light does not seemingly affect the tissues unfavourably.

The individual details of the component parts of the neuron are somewhat finer than by the Cajal method; each element stands out clearly and distinctly, the axons with their collaterals are clear, and the gemmulæ, or buds on the protoplasmic processes, are fully and equally impregnated, and appear in their proper relations to the parent dendrite. There is always some coarse precipitate along the margins of the sections, but rarely the finer one that is so annoying in the older chrome-silver specimens.

The Lesions found in Preparations hardened in Absolute Alcohol.

This first part of the study was conducted on the brains of two rabbits hardened in absolute alcohol, and stained according to the methods of Nissl, and with hæmatoxylin-eosin.

In order to fully appreciate the structure of the pathological nerve cell, stained by the Nissl procedure, we must first glance at the normal cell as seen in the control preparations. The aniline dye does not stain all portions of the cellular protoplasm equally, but leaves clear spaces between others more deeply stained—the chromophilic particles. In the cell body the arrangement of these chromophilic particles is always definite, though varying considerably according to the character of the cell itself. Thus, in the anterior horns of the spinal cord we find a

net-like arrangement of the stained particles (arkyochromic cells), while those of the cerebral cortex have them mainly arranged in rows (stichochromic cells). Furthermore, these various cells are divided into several main groups by the different relations of the nucleus to the cellular protoplasm. Only two of these groups are ordinarily to be seen in the cortex, mainly cytochromic cells, or nerve bodies having a nucleus approximately the size of a leucocyte, and with a very small amount of stained protoplasm surrounding it, and somatochromic cells having a nucleus of moderate size surrounded by a considerable body of protoplasmic substance with a definite contour.

With the cytochromic cells we have but little interest—the main body of the cortical cells being somatochromic, and of the stichochromic variety.

Furthermore, the nuclei present variations in their size, contents, refraction, and receptivity to the absorption of the aniline dyes. The arrangement of the contents of the nucleus also varies considerably. In some we find only a single well-defined nucleolus, the edges smooth and rounded, surrounded by very fine dust-like grains, all deeply stained by the dye, the whole imbedded in a transparent karyoplasma that does not take up any of the aniline. Others have two or three nucleolar particles; seldom more than this number, all smooth, and situated in the several quarters of the nuclear circle, imbedded amidst dust-like grains of a finer or coarser character.

Besides these several varieties, a third exists, in which the nucleolus is less distinct than in the others, and the nuclear dust is coarser than in the other varieties. All these forms present little difference in their staining qualities, the amount of dye taken up outside the nucleoli and dust is very small, and the nucleus, in consequence, is invariably refractile and light coloured. The fourth variety of nucleus is differentiated by the variation in the receptivity of the staining fluid. It absorbs a little more of the dye, sufficient to colour it slightly, but the numbers of these cells in the healthy brain is minimal, and for the practical examination they may be excluded.

Histology of the Absolute Alcohol Sections from the Alcoholic Brain.

The two rabbits of this series have to be considered separately, on account of slight anatomical differences.

Vessels.—The capillaries and intermediary vessels in the control specimens have a smooth, regular contour; the nuclei in the walls are sharply stained, and take up an equal quantity of the colouring matter, and the cell structure between the nuclear points is free from a suspicion of colour (eosin specimens do, however, have a slight staining of the capillary wall with the dye). The perivascular spaces are narrow but distinct. In rabbit No. 10, the smallest arteries have an altogether different appearance from the control sections. The walls of many of them are irregularly shrunken, at intervals almost botryoidal in appearance, and the nuclei of the vascular walls, while not increased in numbers, are swollen, absorb more of the aniline stain, and project considerably into the lumen of the canal. The perivascular space is larger than in the control preparations, and the hyaline membrane separating it from the cerebral substance is a little more definite than normal. As we progress upward in the scale of size, the changes of an abnormal character do not increase greatly, except that the vascular walls are slightly thickened, while the irregularities in their boundaries are gradually lost; but, on the other hand, the nuclei, especially those of the outer layer of the vessel, are somewhat multiplied—at times, though rarely, sufficiently so to cover the exterior of the vessel with rather dense aggregations of round nuclei, through which the proper structure of the canal is seen with difficulty. The peri-vascular spaces around these vessels are larger than usual, and the cerebral edge of the space stains more deeply than normal. Occasionally there are a few grains of hæmatoidin *débris* within the space; more commonly it is vacant. On transverse section, the walls of these vessels appear to be a little thickened—the change affecting the intima more than the media or externa. Very few of the arteries show any very distinct changes. The larger veins

do not show any alterations from the normal. Four or five miliary hæmorrhages, into the sheaths of the vessels, were noted in the several sections, all near the pial margin. Altogether, few definite lesions could be determined in the blood vessels—the perivascular dilatations and hæmorrhages excepted.

In rabbit No. 10, the vast majority of the nuclei of the somatochrome cells of the cortex show similar alterations and departures from the standard established by our control preparations. The most prominent of these changes lies in the central nucleolar figure, for in the place of the smoothly appearing dot, in or near the centre of the ring, it now appears roughened, spongy, or even with elongated projections from its surface (compare photos. 1 and 2). Not only is the nucleolus roughened, but it is also considerably enlarged, occupying now from an eighth to a sixth of the interior of the nucleus—its projections extending to the periphery of the nuclear ring. Around these nucleolar figures are grouped a small number of nuclear dust particles resembling an irregular circle, but the remaining portions of the nucleus are comparatively free from the presence of molecular particles, in contrast to the control preparations. In the clear karyoplasma there is a decided tendency to take up more than normal of the aniline stain; so marked does this tendency become at times that the contents of the nucleus are rendered indistinct, and the body becomes much less refractile than natural.

The above description applies exclusively to the mononucleolar variety of nuclei. The varieties showing two or more nuclei have nearly disappeared, and it is highly probable that the largest masses of spongy nucleoli of the altered nuclei are formed by an agglutination of these chromophilic masses, for indeed in not a few of the cells enlarged and swollen nucleolar particles are seen approaching each other in some portion of the nucleus.

Hardly any nuclei are found differing widely from these two pathological varieties, though not an inconsiderable number of the normally disposed nuclei are seen here and there in the field, the proportion, however, being inconsiderable.

To recapitulate, we find alterations of the nuclei chiefly in the disposition, size, and irregularity of contour of the nucleolar chromophilic particles.

Rabbit No. 39.—The blood vessels show the same condition as in rabbit 10, only not so accentuated. The botryoidal appearance is not so marked. The nuclei of the intermediary vessels are only multiplied to a small extent, an alteration that extends to the smaller veins. In one small artery I detected a distinct aneurysmal dilatation.

All the nerve cells take up a larger quantity of aniline dye than normal, the chromophile masses of the protoplasm are not distinct, the nuclei are nearly all mononucleolar, and the nuclear substance stains a deeper hue than in the control. The nucleoli are comparatively seldom swollen or enlarged, here and there one is seen, perhaps an average of one in fifteen cells, but in many others there is a tendency to be out of proportion in size to the surrounding ones. The nuclear molecules are fine, while the substance itself takes up an unusual quantity of the stain.

A few sections of the cortex of this rabbit were stained by the Weigert-hæmatoxylin method, but I could detect no variation from the normal among the fibres of the coarser bundles of nerve filaments. The finer intercellular fibres were not very numerously stained.

The Normal Structure of the Rabbit's Cortex as seen with the Silver Method.

The arrangement of the nerve cells in the cortex of the rabbit's brain differs little from that of other mammalia. The structure consists of three indistinct layers of cells, with beneath them a rather thin mass of white matter. Viewed with a low power there is but one distinct cellular layer. In the outer or molecular lamina are situated a minimal number of small fusiform and angular cells whose axis-cylinder processes seem to course parallel with the surface of the brain. Beneath this region lie bicornuate, small pyramidal, elliptical, and irregular cells, their axons nearly all turning downward. Beneath these cells lie others of approximately pyramidal

form, but of a larger size than those immediately superjacent to them, and mingled among them are a few irregular cells. All the neuraxons from this region turn downward into the radial bands of fibres. The apical dendrites of nearly all the large pyramidal cells reach past the middle of the molecular layer, and by division form an intricate meshwork of protoplasmic extensions beneath the pial limit. The dendrites of the irregular cells spread out to a greater extent laterally than vertically, but without definite course.

Beneath the pyramidal cells having long apical processes, and bordering on the white medullated fibre layer, lie cells of quite a number of forms, mainly irregularly, pyramidal and angular. The stronger processes of these cells are upturned, while the axons are down turned. Situated between these cells and far more numerous, are small irregular cells, with protoplasmic extensions outstretched in all directions. Their neuraxons are all turned towards the surface of the brain, and on their way thither they give off numerous collaterals, and finally reach the tangential band beneath the pial surface, among whose fibres they are eventually lost.

All the pyramidal cells of the cortex of whatever kind, and very many of the irregular and angular ones have, coming off from their protoplasmic extensions, short rectangular or oblique projections or *gemmulæ*, that have been so universally found by the silver method in the brains of all animals, as not to admit of the possibility of a doubt that they are present during life and are not simply artifacts as was once supposed (Koelliker), indeed they have been demonstrated in particularly fine Nissl preparations (Lenhossék), and are undoubtedly a factor of considerable importance in the economy of the nerve cell, though their function at the present time is somewhat in doubt. On the apical processes of both the long and short pyramidal cells, the gemmulæ are usually simply roughenings on the process near the basal end, higher up they lengthen out, and have a bead-like ending. On the thinner branches of the cells, especially those that come off from the basal extremity, they are longer, thinner, and are arranged with considerable

regularity, and give to these dendrites something of a feathered appearance.

Besides the gemmulæ, the nerve cells have, upon their branches, a small, though very variable number of varicosities. On the thicker portion of the apical processes, these varicosities are never present, but in the finer dendrites they are here and there to be met with, particularly at the branchings of the stems. These varicosities must be held to be normal, as they are to be demonstrated in methylene blue as well as in silver preparations.

With the Purkinje cells of the cerebellum, the gemmulæ contribute greatly to the actual outspread of the dendrons. They are arranged with great regularity, and endow the extensions with more of a feathered appearance than those of the cerebrum, but differ from those belonging to the pyramidal cells anatomically, in that they have few of the beaded endings peculiar to these psychical cells. Knots upon the extensions of the stems of the Purkinje cells are frequent, are of small size, and give the branches an uneven appearance.

Along the outer edge of the cortex numerous forms of support—neuroglia cells, such as have been described by Retzius and others, are seen, but lower, among the nerve cells, the glia elements are but sparingly stained. Few of them belong to the long-rayed Golgi type, but are replaced by a cell, with quite large body, and short but thick, and knotty arms. These cells are always to be seen in the immediate vicinity of an intermediary blood vessel, which they frequently surround, and send off to its hyaline sheath one or more thick processes that terminate in knob-like endings. In disease, unlike some other varieties of neuroglia, they atrophy and disappear.

The Pathological Histology of the Rabbit's Cortex in Chronic Alcoholism.

The material for this portion of the study comprised the brains of three rabbits preserved in Müller's fluid. The lesions found in all the specimens were identical.

In formulating any description of the pathological changes found in chronic alcoholism, we should always have prominently before our mental vision, the fact that the normal characteristics of the cortical cells as we see them by any of the more usual methods of preparation, vary within certain fixed limits, and that these must be overstepped before we can say with certainty that we have before us an abnormal condition. By any method of staining with the chrome-silver salts this is perhaps more strikingly the case than by other methods, and the observer must be constantly upon his guard against deceptions into which he may very innocently fall.

A portion of the uncertainty of the silver method we have been able to obviate from the fact that the new mode of staining we have pursued is more fixed and reliable for the adult brain than any other treatment we have hitherto tried; truly a comparison of many scores of normal cells in the control slides show little or no variations in the cell beyond those inherent to the several varieties.

The intrinsic variations in the formation of the neuron are for the practical purposes of this examination reduced to the normal inequalities of the varicosities in the construction of the dendrons. These inequalities in the cortical dendrons are of comparatively infrequent occurrence, in contrast to those of certain of the cells of the basal ganglia, and belong either to the finer dendrites of the irregular cells, or to the points of division of the dendrites, and are then usually of triangular form and small size, and may readily be excluded in an examination for pathological lesions.

Besides the swellings in the course of the dendrons, we have always to be on the look out to exclude certain processes of the neuroglia cells that traverse long distances in the cortex, and exhibit a pearl-string swelling in the course of the fibre. These nodal formations lie at perfectly regular distances one from another, and present the appearance of a double cone of small size. It is only when these fibres are detached from the cell body that they can give occasion to error. Another source of trouble to the observer is the

occasional deposit of rounded masses of precipitated silver on the dendrons or cell body, but this may usually be cleared away without difficulty by a careful examination of the edges of the questionable mass with strong transmitted light, when the irregular structure of the precipitate and serrated edges will at once appear. Still another source of error is the fracturing of the protoplasmic processes by the sharp edge of the knife (see fig. 4), the potash salt rendering the protoplasm brittle, but once aware of the difficulty it is easy to eliminate it as a source of error.

After all possible allowances have been made for artefacts and physiological variations, together with inequalities in the staining process; there remain a large number of cells in the alcoholic brains that are distinctly abnormal. The principal lesions to which we would call attention are a distinct diminution in size, a shrinkage of a vast majority of all the cortical cells, disappearance of the gemmulæ, certain swellings in the dendritic processes; and roughening of the stronger processes, and to some extent of the cell body.

We have not succeeded in staining the fusiform cells satisfactorily in the alcoholic rabbit's brain, but with this exception the cortical cells of all the layers may in chronic alcoholism be considered to be involved in a process of retrogressive metamorphosis, some in greater, some in less degree, but all are implicated to some extent. The small cells of the outer layer show degeneration, the pyramidal cells, particularly the long ones, show the same changes in greater intensity, the lowermost pyramidal and irregular cells are equally involved. Fortunately the pyramidal cells, from their very fixed character and larger size, are more easily studied than the others, and our principal attention has been directed toward them.

It is extremely difficult to determine in the silver preparations the approximate proportion of cells that are normal, and cells that may definitely be held to be abnormal, from the fact that an extremely small proportion of the total number of cells in all the layers are at any time stained. In some of the preparations from the same brain but one in a dozen cells show any definite change, while in other

sections nearly all the cells are altered. It would perhaps be fairly safe to conclude that one out of every three or four cells in the brains of the three alcoholic rabbits we examined show departures from the normal in some form, though the percentage is probably higher. Another source of error in estimating the numbers of diseased cells arises from the circumstance that we can form no idea whether the silver salt preferably impregnates the normal or abnormal cells, for even in histological preparations no one has advanced any plausible theory why the silver elects here and there a cell to impregnate, and leaves the others perfectly untouched by the salt. Golgi (*Berl. Klin. Woch.*, No. 14, 1894) himself opened the question as to the capability of the diseased cell receiving the silver stain, but could only answer it to the extent that even much degenerated cells were impregnated.

A careful comparison of a control preparation, and one from an alcoholic rabbit will show the following differences: the cell bodies and main processes are more even and smoother, the dendrites are broader, the gemmulæ are longer and more numerous, thicker, more feathery, apparently spreading over more lateral surface, and are very regular in appearance, while in the alcoholic there is a gap here and there as if some of them had fallen off, or had disappeared. Indeed, the difference between the two cells may shortly be expressed in that the whole aspect of the normal cell is stouter than its pathological fellow.

Among the cellular layers—and we take the pyramidal cells of all sizes as our chief exponent—we find a vast number of the cells which show upon their protoplasmic extensions one or more swellings of a rounded or elliptical form (figs. 3, 6, 7). Some of these tumefactions are very small, and only to be noticed after close search, while others are so large as to immediately attract attention, and excite suspicion that they are artefacts, and indeed it was only after a thorough examination by means of the electric light that we were able to disabuse ourselves of the idea that they were not artificial productions (fig. 5). Under the intense illumination of the electric lamp they appeared as uniform reddish bodies divided longitudinally (under high magnifica-

tion) by a series of fine lines converging upon the substance of the unthickened portion of the dendrite. The number of the swellings upon an implicated dendrite may vary very considerably; a dendron may be almost wholly filled with the swellings, at other times there may be only one or two, of either large or small size (figs. 3, 4, 7, 8, 9).

The process of tumefaction always appears to begin at or near the fine free extremity of the dendron, be it the extremity of the main apical process or one of its collateral branches, and not infrequently the free termination of the dendron is seen to be somewhat swollen before any other portion of the cell is involved. Looking over the preparations a little closer, we find numerous cells with moniliform swellings stretched along the finer apical processes (figs. 3, 4, 6, 8, 9, 10), until a number are seen with the whole of the apical processes thickly studded with the bead-like swellings. Somewhat remarkable to relate, the basal dendrons are only exceptionally involved in the tumefactive process, though they do not escape alteration when the cell corpus eventually begins to shrink (figs. 10 and 12).

An essential accompaniment of the tumefaction of the protoplasm of the dendron is the loss of the gemmulæ. The smallest swelling causes along its edges an evident decrease in the number of the buds (figs. 4, 6, 7). After the swelling has increased to a considerable degree all disappear, while in the intermediary stages every step toward decrease and disappearance may be found. Between the points of swelling they are retained, but are often seen to be thinner and situated at more irregular intervals than normally. When long thickenings occur in the dendritic stems the buds are invariably lost. The last of all the gemmulæ to disappear are the thicker branched ones that project at intervals some little distance beyond the others, and at times, though very rarely, they may still be seen clinging to the sides of some not far advanced swelling.

A very accurate comparison, so far as outward appearances go, may be made between the moniliform swellings of the dendritic processes of the nerve cells, and the swellings on the branches of some species of the oak tree caused by a

parasitic insect. Both have the same knots and swellings on their branches, even to the falling off of the finer lateral twigs on the tree when the disease has advanced to a considerable extent, and both may go on to produce a shrinkage of the substance, finally ending equally in decay and death to the plant and cell.

As the arterial changes found were comparatively unimportant in their character it would be difficult to ascribe this widely extended and curious process of tumefaction of the dendrites and disappearance of the gemmulæ to nutritive changes solely from a defective supply of nourishment to the nerve cell, though truly the definite enlargement of the peri-vascular spaces would indicate that there had been some previous disturbance in the circulation of the lymph currents of the cortex. It would seem much more plausible to attribute the lesions to the direct action of the poison upon the protoplasm, though why it should take this peculiar form is difficult to determine. The alterations can hardly be of fatty nature, as the bodies of the unstained cells show nothing of this nature beyond the ordinary amount of yellow pigment granules which in the rabbit is minimal.

It would appear from our specimens that the moniliform swellings of the dendrites and the loss of the lateral buds was the first step in a pathological process, which eventually was to end in the partial or complete disintegration of the cell structures, and with it the annihilation of the nervous forces produced by the cell, the latter probably preceding anything like cellular death, for clinically we frequently see almost complete dementia alcoholica, when according to the ordinary methods of staining, but comparatively few of the nerve cells show characteristic signs of degeneration.

Only a few of the nerve structures of the rabbit's brain give any evidences of deterioration of the substance of the corpus, beyond those already described, though occasionally a neuron may be found which exhibits a more advanced degree of degeneration. Then the processes immediately adjacent to the body are roughened and seamed like the coarse bark of a tree; the staining of the altered protoplasmic substance is irregular, the cell body is irregularly

shrunken, the basal dendrites no longer stretch over wide areas like their normal fellows, but have dwindled, the gemmulæ are lost, and the processes are not stained for any distance from the corpus of the cell, but soon end in needle-like points (fig. 12). Some of these cells might even be described as being corrugated, from the very roughened appearance of their exterior.

The nuclei of the nerve cells are seldom to be seen in silver preparations; occasionally a vacuole, occupying the spot where the nucleus usually lies, can be determined, and in its centre a black dot, probably the nucleolus, may be found if a careful search be made for it, but no details of the intimate structure can be determined. A very few of the sections exhibit a process we have thought possibly may arise from defects in the impregnation, though on any ground it is difficult to account for. The dendrons and axons of the neuron are nearly perfect, while the cell body looks as if a portion of its substance had been scooped out down to the region of the nucleus. Such a cell is pictured in fig. 11.

The axis-cylinders of all the cells of the cortex were made the object of careful study, but no morbid alteration could be made out in them; but this continuance of the axon, when the other portions of the cell structures are far degenerated, is one of the peculiar features of the neuron which has before attracted attention, especially by Golgi (*loc. cit.*), who found that the axon resists to a greater extent degenerative processes than the dendrons, and may be found intact when the cell is reduced to a mere stump. In all our preparations very numerous axis-cylinders were stained, and could be traced from their commencement at or near the basal end of the cell, downward into the white layers, or upward toward the uppermost layer of tangential fibres; but no trace of unusual varicosity or break in their continuity could be discovered. The collaterals oft-times could be followed considerable distances, occasionally to their free endings, but nothing abnormal could be seen. Cells in an advanced stage of degeneration had as perfect axis-cylinders as normal ones, and could be traced equally long distances.

Cortex of the Cerebellum.

The Golgi cells and the Korbzellen of Koelliker stained only in inconsiderable numbers, and but few of those impregnated showed changes comparable to those of the cerebral cells. The Purkinje cells, on the other hand, were luxuriantly tinged, sometimes an entire row being blackened and exquisitely brought out. A better comparison could therefore be made in these preparations between the numbers of degenerated and undegenerated cells—the proportion now being, when an entire row was taken, one degenerated to three normal ones.

Practically the same alterations were found with the Purkinje as with the pyramidal cells, though the changes were more striking from their relative intensity. The loss of the lateral buds, from their greater luxuriance, was extremely striking; they disappear from the dendritic branches as an entirety, and on a considerably degenerated nerve body not a single one can be found (figs. 15, 16, 17).

The stems of the dendrites also undergo atrophy, and in great measure disappear; only thick stems are now seen arising from the corpus of the cell, which give off a number of short, stumpy branches, thickly studded with knotty projections in the place of the long feathery dendrites and their terminal twigs (fig. 14). These twisted and shortened stems have numerous swellings in the longitudinal direction of their protoplasm. Those that still retain something of the form of a dendrite have numerous irregular swellings in the stems of the finer twigs; the gemmulæ are almost universally lost, and the whole appearance is as if the dendrites had become gnarled and knotted by the action of some parasitic insect (fig. 15). The cells that are least implicated have the rounded and longitudinal swellings along their finer stems, and the buds along the margin are fewer than elsewhere.

The degeneration of the Purkinje cell, while having the same general characteristics as the cell degeneration in the cerebrum, is much more striking, and there is no possibility

of mistaking the process for anything else than it actually is—a degeneration of the cellular structures.

Neither in the cerebrum nor cerebellum is there any pronounced change in the support elements. The Deiter's cells stain in neither region with any frequency, while the large-bodied vascular cells do not seem to take on any alterative action, clinging to and surrounding the vessels after the usual manner.

Before concluding this paper let us glance for a moment at the clinical picture of chronic alcoholism in order to see in what manner the pathological lesions and the symptoms correspond with one another. The epiphenomena of chronic alcoholism are so numerous that it will be difficult to do more than sketch the most prominent symptoms: First come the sensory disorders, the exaggeration of the sensibility of the skin, the anæsthetic symptoms, the ocular and auditory troubles; then follow the tremor, the tremulous articulation, the loss of tone in the muscular system, the tottering gait, the occasional paretic attacks, the inco-ordination, and lastly the intellectual disorders, the loss of moral force, the weakening of the mental faculties, the falsification of the judgment, the slowness of memory, and finally the dementia, which is seldom complete.

The first of these periods would correspond to the beginning of the vascular disorders, when the nerve cells, irritated by the presence of a poisonous stimulus and an insufficient supply of proper nourishment, overact for the time, and then as nutriment is still withheld from them, altered metabolism results. The beginning swellings of the dendrites of the sensori-motor region is marked by anæsthetic and paræsthetic symptoms, those of the purer sensory regions by visual and ocular troubles and some amnesia, especially for recent events, or in other words, cells that have the function of evolving thought cannot work properly, and defective memory results. Later, as the motor cells become more and more involved and nuclear changes begin, continuous tremor becomes apparent, the muscles no longer co-ordinate perfectly, unless for a moment under the direct influence of

the will; still later, when a portion of the cell structures become highly degenerated, and the altered cells have become more numerous, the already tottering will power becomes less and less active, judgment and memory fail, and when the degenerative process has far advanced an incomplete dementia is the final result.

In conclusion, we do not consider the fact that only a portion of the cells of the cerebrum are involved in the degenerative process militates aught against the entire conception of the pathological entity. The nerve elements of the brain are intricately united one with another by means of their axons and collaterals, and are not able to function completely unless the normal relations one to another are preserved. A lesion in one cell will induce disorders in the function of two or more cells not involved by any morbid change, the intricate system of collaterals issuing from one cell influencing directly the impressions and nervous forces arising from many others, and in this way a widespread disordered action of large numbers of the cerebral cells may be the result of disease in a comparatively few elements.

In the human being we have found the same degenerative process present as in the rabbit, varying only in degree, in some more intense, in others less so; but as the pathological lesions in chronic alcoholism in the human subject will form the basis of a subsequent paper, it will suffice here to say that they are practically identical with those in the rabbit.

We do not for an instant consider these lesions of the neuron peculiar to the effect of alcohol, but regard them as capable of being reproduced by any irritant drug or bacterial toxic product circulating in the blood, and acting for a considerable time on the living protoplasm of the nerve cell.

One point is made definite by this study—the fact that the alcohol which was supposed to be the least deleterious of all the series, exerts a very definite and destructive effect upon the nerve cell.

EXPLANATION OF THE PHOTOGRAPHS AND DRAWINGS.

Photos.

FIG. 1.—Nuclei of normal pyramidal cells of the rabbit's cortex, showing the nucleoli smooth and sharply defined. (× 1100.)

FIG. 2.—Nuclei of the pyramidal cells of the alcoholic rabbit's cortex, showing the nucleoli roughened and enlarged. The outline of the nucleus is also more irregular than in the normal cell. Rabbit No. 10. (× 1100.)

FIG. 3.—Beginning moniliform swelling of the apical process of a small pyramidal cell. Rabbit No. 42. (× 275.)

FIG. 4.—Moniliform swellings in detached portions of apical dendrites. Rabbit No. 42. (× 275.)

FIG. 5.—Large swelling in the apical process of a pyramidal nerve cell. The broken appearance of the dendrons in the figure is artificial and made by the microtome knife. Rabbit No. 41. (× 265.) Micro-photographs by Dr. A. G. Hoen.

Drawings.

FIG. 6.—Small pyramidal nerve cell from the outer portion of the second layer, with normal body and basal and apical dendrites, but with one branch from the apical dendrite showing moniliform swellings. The gemmulæ have disappeared wherever a swelling has begun. (× 560, enlarged.)

FIG. 7.—Small irregular nerve-cell from the outer portion of the second cell layer with advanced moniliform swellings of all the principal dendrites, and roughening of the cell corpus. (× 560, enlarged.)

FIGS. 8, 9.—Advanced moniliform tumefaction of apical dendrites. (× 560, enlarged.)

FIG. 10.—An irregular pyramidal cell from the deeper portion of the second layer in an advanced state of degeneration.

FIG. 11.—Pyramidal cell from the deeper portion of the second layer with slight roughening of the apical process and normal dendrites. The corpus of the cell is excavated from its basal edge to the further limit of the nucleus.

FIG. 12.—A medium-sized pyramidal cell showing disappearance of the long dendrites and great roughening of the thicker stems, as well as complete loss of the gemmulæ. The cell body has a very irregular contour and is shrunken. The axon is intact. (× 560, enlarged.)

FIG. 13.—Terminal portion of a dendrite of a Purkinje cell showing the normal arrangement of the gemmulæ. From a control preparation.

FIG. 14.—Portion of a process of a Purkinje cell from near the corpus. The longer branches and gemmulæ have entirely disappeared, leaving an irregular knotted stump. (× 560, enlarged.)

FIGS. 15, 16, 17.—Terminal branches of the dendrites of Purkinje cells, showing the irregular swellings of the stems, loss of the gemmulæ, as well as the finer branches. Rabbits Nos. 6 and 41. (× 560, enlarged.)

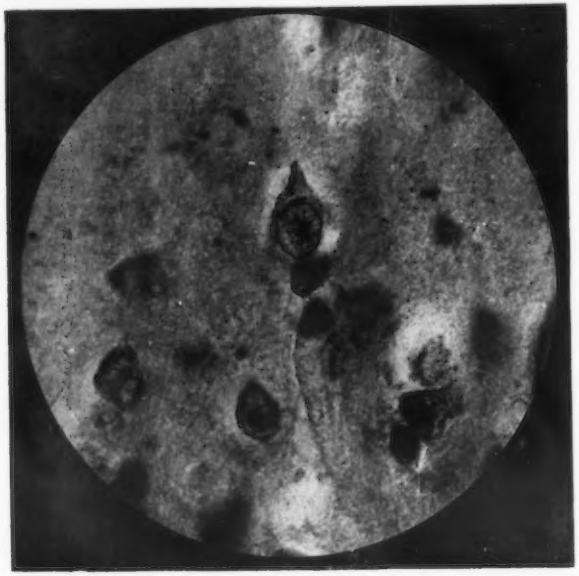


Fig. 2.

Fig. 1.

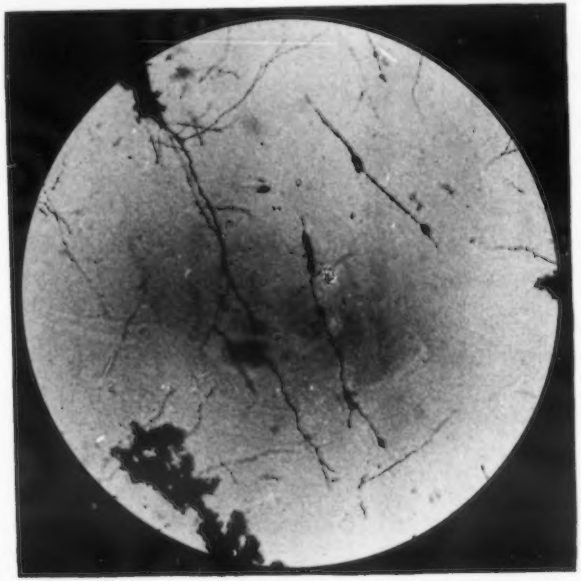


FIG. 4.

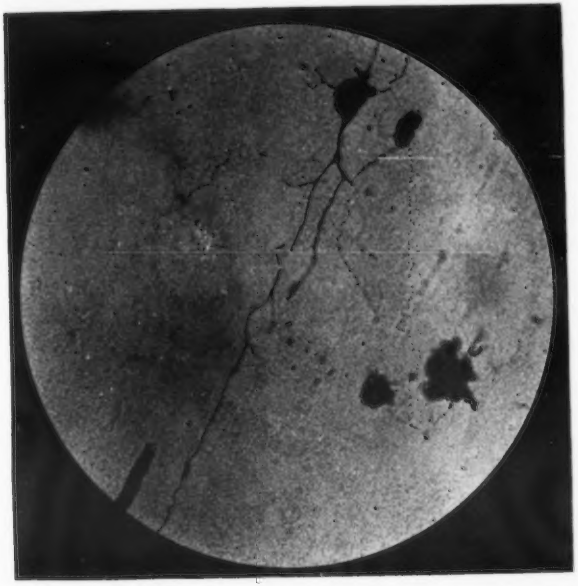
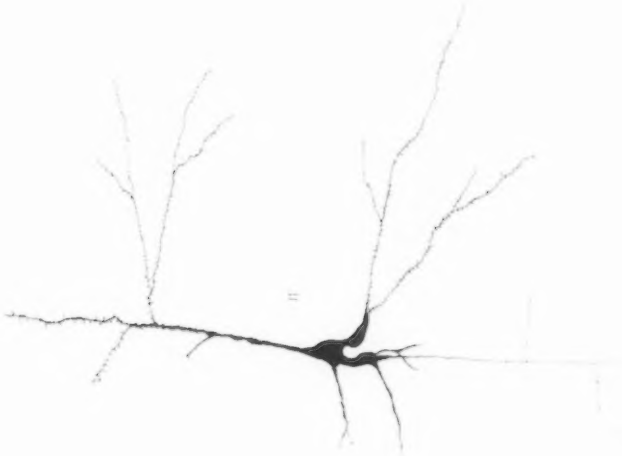


FIG. 3.



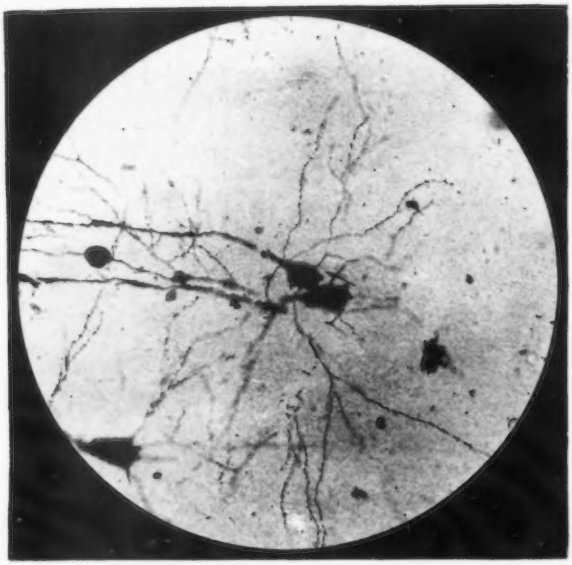
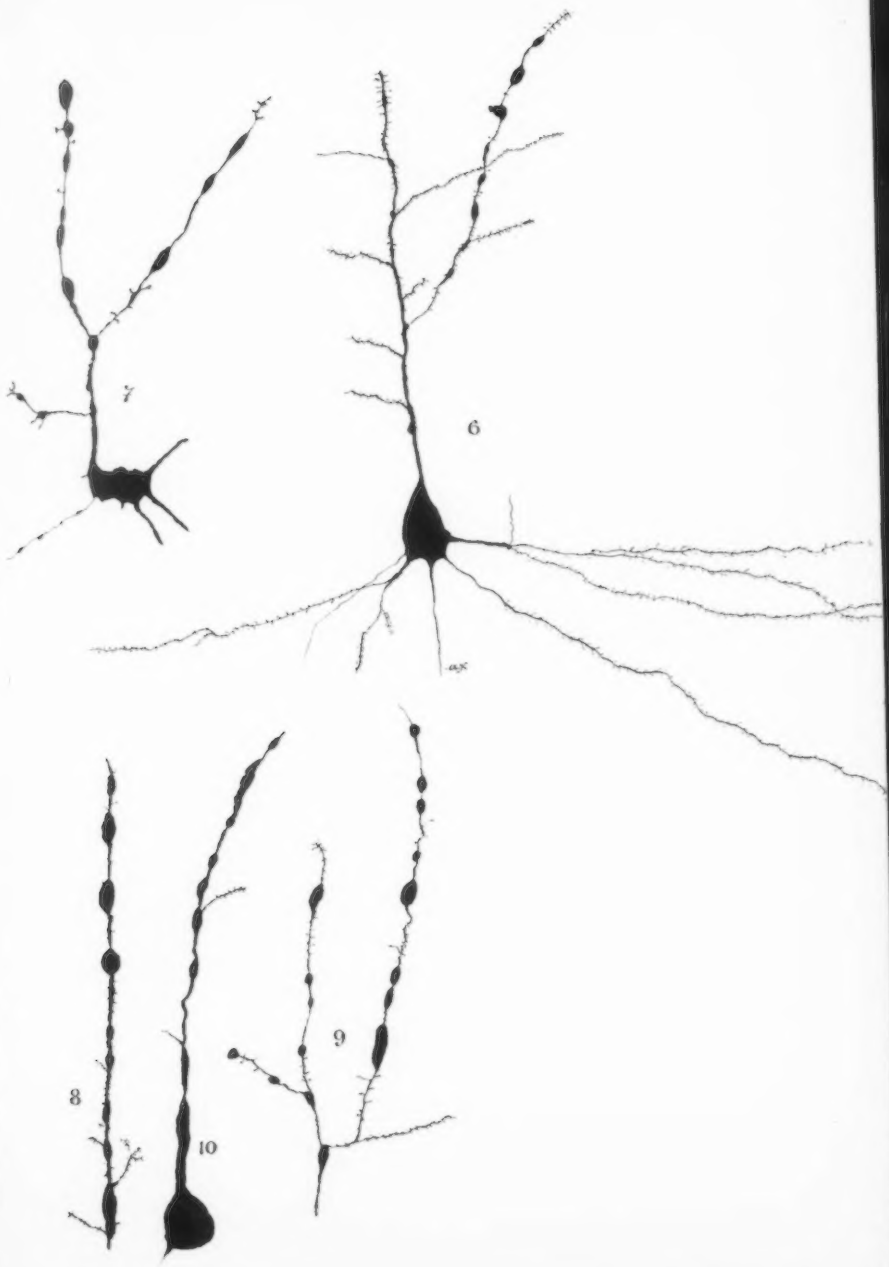


Fig. 5.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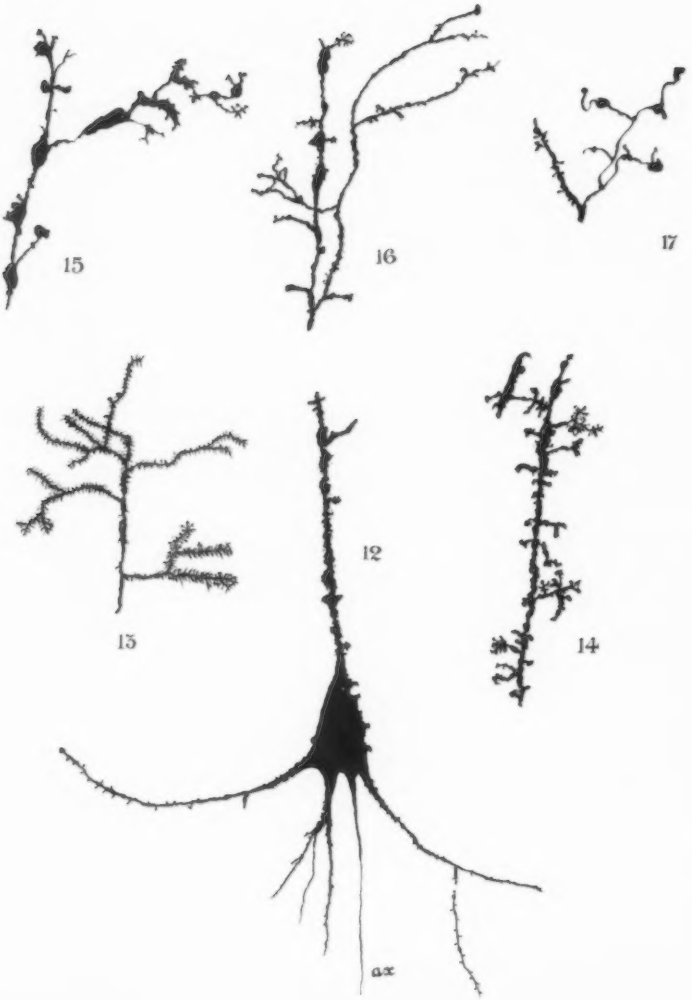
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THE FUNCTIONS OF THE FRONTAL LOBES.

BY L. BIANCHI, M.D.

Professor, and Director of the Clinique of Mental Diseases, in the University of Naples.

[*Translated from the original MS. by A. de Watteville.*]

THE experimental problem raised by the question we are about to discuss is of recent origin, and aroused the attention of observers soon after the discovery of motor and sensory centres. On electrical examination of the cortical surface, the anterior frontal lobes were found not to be excitable; and whilst ablation of other areas was followed by more or less evident loss of movement or sensation, that of the præ-frontal region gave rise to but slight, or doubtful, disturbances.

These negative results are the cause of the small amount of attention paid to the physiology of the frontal lobes, as compared with the activity displayed in experimentation on the motor zone. But I hope that the experiments I am going to describe, conducted on those more rigorous lines adopted by our modern science, will, to a certain extent, make up for the deficiencies of the previous results. I think, however, that we may pass over the researches of Müller, Longet, Vulpian, Lussana, and others; they vainly strove to transfer into the domain of the positive school, the results of the reaction against Gall's organology, such as that intellective activities (images, memories, ideas) are not localised in any particular areas of the cortex.

The classical work of Hitzig[1] afforded a more secure starting point, and by new methods opened a new phase in the evolution of our knowledge of præfrontal functions. He observes that anatomically the development of the frontal lobes is found to keep pace with that of the intellect, being less in the cat, for instance, than in the dog, and still more so than in the monkey; hence the author concluded, naturally enough, that they are really the seat of the highest intellectual faculties. But Hitzig's experimental results did not confirm this very plausible argument. His dogs never showed intellective disorders, either after superficial lesions, or after complete extirpation, of the frontal lobes.

Ferrier[2] next attempted to solve the problem, on the strength of a more abundant experimental material. He assumes that all in front of the sigmoid gyrus represents the frontal lobes in dogs and cats; in monkeys, what is in front of the centre for the movements of the eyes and face and the opening of the lids and dilatation of the pupils. Electrical excitation of this region has always been negative, except in one monkey, in which ocular movements were observed.

Mutilation of this region gives negative results in dogs. Monkeys (three in number) showed no alteration in their movements, sensations, instincts, feelings, or emotions. There was, however, some alteration in the temper and behaviour of these animals, says Ferrier, though it is difficult to specify its nature. They took less interest in their surroundings; showed less of their natural inquisitiveness; were apathetic, somnolent, responded only to momentary impressions, and had lost the power of attentive observation. Ferrier attributed motor functions to the frontal lobes on account of their connection with the corpus striatum; they are inhibitory centres, whose activity is exerted on influencing the actual motor centres. Inhibition thus understood is nothing but attention, which is thus to be considered as a function of movement. Hence his conclusion that the frontal lobes are the centres of attention.

[1] "Untersuchungen ueber das Gehirn," 1874.
[2] "The Functions of the Brain," London, 1876.

Ten years later, in his second edition, this author comes to nearly identical conclusions. He confirms the absence of electrical excitability of the part of the frontal lobe placed anteriorly to the eye and head centre (No. 12 of his diagram). He relates new experiments on the destruction of the præfrontal region, remarking, concerning one of them, that notwithstanding the bilateral lesion, there is complete absence of symptoms, either motor or sensory. The animal feels contact over the whole body, the special senses are intact, and the movements of the head and eyes are completely preserved.

Extirpation of the anterior extremity of the frontal lobe having produced the same deviation of the head and eyes as that of the region immediately behind (marked 12 in the diagram), Ferrier was led to consider the two as essentially the same functionally, viz., as motor of the head and eyes.

It is impossible to anyone familiar with these experiments not to look upon this conclusion as premature. Ferrier was too much impressed by the phenomena observed immediately after the operation. We now know how many errors are committed by this cause, as Goltz pointed out long ago. Nor does the argument derive much support from the fact of the anatomical relationships shown to exist between the frontal lobe and the pons and medulla by the microscopical examination of brains thus operated upon. For nothing proves that a centre of higher function cannot have relations with the pons, cerebellum and medulla; and that a degenerated tract traced through the internal or mesial part of the internal capsule and pes cerebri must necessarily subserve movements. And even if the frontal lobe were motor, it does not follow that it must be considered as the centre for the head and eyes, as are the roots of the superior and middle frontal convolutions.

This criticism is the more founded, as we find that in one of the experiments, destruction of the frontal lobes gave rise to no symptom, though secondary degeneration was traced through the internal capsule.

Even in this second series of experiments, however, Ferrier

succeeded in showing evidence of a mental deterioration. The monkeys, after the operation, had lost the power of attentive observation. But this fact was much less evident when, through antiseptic precautions, the animals were enabled to survive for a long time. At any rate, as the author says, this fact was more apparent than real. But, adds Ferrier, the frontal lobes are not the seat of intelligence; this would be an absurd statement, since intellect and will have no distinct localisation, but are the resultant of the functions of the whole cortex, and are correlated to the harmonious development of its various portions.

Thus Ferrier conciliates his first hypothesis, that the frontal lobes are the centre of attention with the views promulgated in his second edition, according to which it is so because it is a motor centre, and makes part of the motor zone.

Moolmann's researches added nothing to the doctrine propounded by Goltz, in whose laboratory his researches were conducted. Here are his conclusions: disorders consecutive to destruction of one frontal lobe do not differ as to persistency from that of any other part of the cortex; partial destruction of the frontal lobes causes but insignificant symptoms. When both frontal lobes are completely removed, the consecutive disturbances are the same as those observed after ablation of the parietal lobes, which means, according to Goltz, of any other part of the cortex.[1]

Goltz[2] so far recognised that the animals deprived of their anterior lobes, without being actually paralysed, cannot bring into play the complex processes of innervation necessary to the normal performance of actions, and lose their power of voluntarily moderating their bulbar and spinal reflexes.

Hitzig returns to the subject, and by new experiments on dogs, combats Munk's theory, and asserts that ablation of the frontal lobes is never followed by any motor disturbance.[3]

[1] *Pflüger's Archiv*, Bd. xx., 1879.

[2] *Ibid.*, vol. xxxiv.

[3] "Achte Wanderversammlung d.S.W.D. Neurologen," *Neurol. Centralbl.*, 1883.

We now come to Munk's researches, which were favourably received in Germany, with reference not only to frontal functions, but also to the sensorimotor localisations of the cortex generally. He used dogs and monkeys. Here is a summary of what he says of a dog: Three to five days after a unilateral operation the animal is as lively as before. It manifests no disorder as to vision and hearing under any test or circumstance. It sees, hears, tastes, understands when called. Internal sensations are normal; likewise general sensation and intelligence. A dog thus mutilated cannot be distinguished from a normal animal. It walks, runs, jumps, moves its eyes, ears, tongue and tail as before the operation. It barks, bites, feeds as before. The movements of the trunk alone show some disturbance. If the extirpation has been carried out on the left side, the dog can walk and turn to the right and to the left. But when it turns suddenly it forms an arch, whose convexity is towards the right side, the body is arched on the left, the vertebræ forming a convexity to the right. On running it can describe large curves to either side, but sharp curves are possible to the left only.

Thus far Munk. Extirpation of the left frontal lobe causes nothing but slight trunk paralysis, of long duration, persisting for months. Extirpation of both lobes produced an abnormal arching of the dorso-lumbar spine; especially on walking slowly or rising from the ground the animal displayed this symptom (cat's back), but not on running or jumping. No disorder of intelligence nor of the various forms of sensation were observable, whenever the excision involved the frontal lobe lying in front of the chief frontal sulcus (Hauptstirnfurche). Similar results were obtained in analogous experiments on monkeys. After the direct effects of the operation had passed away, the animals deprived of their frontal lobes lost their smell only when the olfactory lobe had been also injured. The trunk muscles alone were affected; no disorder of sensation, sensibility, nor intellect was noticed. The animals could not bend their dorso-lumbar spine to either side in the performance of the smaller movements. They could not straighten the back,

which remained inflected, with approximation of the anterior and posterior limbs. What Ferrier observed (apathy, somnolence, unrest) is, therefore, attributed by Munk to the effects of the lesion itself, to encephalo-meningitis.

Munk, therefore, looks upon Ferrier's experiments as failures, denies the existence of any loss of attention, perception and thought, and states that there is no failure of that inhibition which Ferrier takes to be the physiological substratum of attention. He found that a monkey so operated was kept, by a mere look or gesture of menace, from touching a much coveted morsel of food placed before it. This monkey used to take a piece of carrot held between the thumb and index, but not between the thumb and middle finger until the index had been drawn back.

Luciani[1] reports only two experiments on dogs which were deprived of one frontal lobe only. In both cases he observed nothing but a paralysis of the dorsal muscles and sensory troubles, with paralysis of the anterior limb, ascribed by him to a lesion of the anterior limb of the sigmoid gyrus.

The researches of Horsley and Schäfer[2] are not at all conclusive, because the mutilation was confined to the anterior third or fourth of the frontal lobes. None of the seven monkeys showed any disturbance of motion, or special sensation nor intellect. These results are wholly contradictory to those of Munk, and partially to those of Ferrier, who in a recent publication,[3] restates his former position without any modification.

Lastly, Grosglik[4] has just published a paper, in which the bibliographical references are incomplete. His experiments have been performed only on dogs, and are therefore undecisive on such a question. He reaches the same conclusion as Munk in so far as the frontal lobes are the common centres of the neck and back, but on the other hand he maintains that the motor troubles of the spine are not persistent.

[1] Luciani e Seppilli, "Le localizzazioni funzionali del cervello," 1885.

[2] *Philosophical Transactions of the Royal Society*, 1888.

[3] "Croonian Lectures," 1890.

[4] Grosglik, "Zur Physiologie der Stirnlappen," *Archiv für Anatomie u. Physiologie*, 1895.

Let us now summarise the various opinions hitherto propounded on this subject:

(1) The præfrontal lobe is the motor centre of the eyes and head on the opposite side; and in consequence of the close relationship between the movements of these parts and attention, it is also the centre for attention.—(Ferrier.)

(2) It is the centre for the highest psychical functions. Destruction of it involves a real decadence of psychical activity.—(Wundt, Hitzig, Bianchi.)

(3) It is part of the so-called "Fühlsphære," and as such is the motor centre of the dorsal muscles. Its highest development is not correlated with that of intellect, but with that of the dorsal musculature.—(Munk, Luciani.)

Such were the various hypotheses concerning the frontal functions when, with wholly unprejudiced mind, I began my own researches in 1888. I have been prosecuting them ever since, making short communications to the Royal Medico-Chirurgical Academy of Naples (1889), to the Society of Italian Alienists (1890),[1] and to the Congress of Internal Medicine in Rome (Oct., 1892). My experiments were made on monkeys and dogs, after the method of free cranial openings and exposure of the lobes, rigorous asepsis and observation of the animals for several months or even years. I took no account of the symptoms during the first week, which must be systematically overlooked in researches on the cortical functions by the method of extensive mutilation.

I used twelve monkeys and six dogs, and never lost an animal immediately after operating, even after total extirpation of both frontal lobes. I lost a monkey in my laboratory at Palermo, from consecutive hæmorrhage; and one last year, a rickety subject, with a thick skull that offered great difficulties in operating. In determining the præfrontal zone, I have not, as did Munk, taken the sulci as landmarks, first, because they never demarcate the physiological areas of the cortex; secondly, because even if such were the case the experimenter cannot always strictly follow their indications.

[1] See abstract in "Untersuchungen z. Naturl. d. Menschen u. d. Thieren," *Moleschott's Arch.*, vol. xiv.

Hence I have not always hit upon the præcentral sulcus in monkeys, nor upon the extremity of the chief frontal sulcus (sigmoid fossa) in dogs.

I have taken as my chief guide the electrical reactions of the frontal region, exploring it repeatedly with an exciter connected with a du Bois Reymond's sledge apparatus. I used to test the strength of current on my tongue, always keeping to the same intensity of sensation, which corresponded to 9 or 10 mm. of the scale. I avoided the use of the galvanic current for fear of trophic alterations in the cortex. On account of the slight individual differences in the motor localisations, especially in the monkey as compared with the dog, I thought it best to carry out the incision two or three millimetres in front of the excitable areas of the arm, face and jaw. I thus almost always in monkeys hit upon the areas, excitation of which produces dilatation of the pupil and lids, and turning away of the head more than of the eyes.

On account of the obliquity, from below upwards, of the Rolandic fissure, it is evident that a greater portion of the upper escaped, than of the middle and lower frontal convolutions.

I did not make any special efforts to spare the olfactory bulb in monkeys, in whose psychical life it plays but a subordinate part; but I always preserved it in dogs. In these animals the line of section fell at less than a millimetre behind the sigmoid fossa; the anterior arm of the sigmoidal convolution being almost always involved.

I shall not dwell upon the results of the electrical excitation of the zone thus removed; they mostly agree with those of other observers; the surface of the frontal area situated in front of the motor area of the head and neck, and of the iris is quite unexcitable with currents of equal strength. I shall only remark that such an excitation of the foot of the upper frontal convolution produced (in monkeys and dogs) not only a slight rotation of the head to the opposite side, but also some lateral displacement of the trunk at the level of the lumbar region.

Conjugate movements of the eyes did not always accompany rotation of the head. Excitation of the inferior area

produced raising of the eyelid along with dilatation of the pupil. In one case this dilatation in the opposite eye could be observed fifteen minutes after the stimulation, which, as I have already stated, I used only in order to fix the line of incision. The latter was not identical in all experiments, on account of individual or of operative differences, hæmorrhage or adynamia compelling me occasionally to hasten the operation. Three to five days after the operation the animals were fit to undergo all the investigations required to determine functional losses.

The following statements summarise the results obtained by unilateral extirpation of the frontal lobe.

(*a*) During the first and second week I often observed, in monkeys oftener than in dogs, rotatory movements, the truncal concavity being towards the mutilated side. No concomitant oculo-motor disturbance is observed. These symptoms do not always occur, and rarely last beyond the second week.

(*b*) Paresis of the opposite arm, not evident in associated movements (climbing, seizing a stick, shaking objects), but obvious in more delicate movements, when the monkey uses the other hand (for picking up a fruit or other food, catching fleas). The animal then uses the paretic only when the other is immobilised. This paresis also disappears after a few days—three weeks at most. In some of the subjects it has not been observable, or only slightly so.

(*c*) Tactile sensibility remained normal, except in a dog, in whose opposite limb it was for a time distinctly diminished, and in a monkey who showed some hyperæsthesia in the opposite ear and face. All the animals scratched themselves equally on both sides, and the monkeys hunted for fleas with their usual and characteristic precision.

(*d*) In one case (a monkey) I found diminution of hearing on the opposite side.

(*e*) Taste appeared normal, as well as smell, so far as could be ascertained in so difficult an investigation.

(*f*) In all cases, monkeys and dogs, I noted visual disturbances. I shall quote the observations made in one instance:—"The opposite eye being closed, the monkey

walks, runs, without ever coming in contact with any of the obstacles placed in his way; he avoids them all without uncertainty. But let him be kept immobile, and his attention be directed on fruit held at from 30 to 40 cm. in front of him; if then a piece of sugar, of which he is extremely fond, be suspended by a thread and gradually brought from the right inwards into his visual field, the animal sees it only when the sugar is nearly in a line with the visual axis. It is only then that the animal perceives it, and seizes it with a rapid and precise movement of the left hand. This does not occur when the right eye is kept closed; as soon as the sugar passes the physiological boundary of the visual field the animal's attention is excited, and the action of seizing the object is instantly performed. This experiment never failed on repetition; the difference between the two eyes is striking. The results were the same a fortnight, as they were a week, after the operation. Generally, these visual disturbances last from two to three weeks.

No perceptible differences were noted in the behaviour or psychical manifestations of animals mutilated on one side only.

I shall now give some details concerning the psychical condition of one dog, and of three monkeys operated bilaterally.

(*a*) Dog whose two anterior lobes were removed on April 16 and on May 16 respectively. The sensory and motor functions of the limbs remained normal. On June 21 the following notes were taken:—As soon as the door of the room in which it is confined is opened, the animal walks out. It goes with eyes downcast, and head bent down, almost touching the floor with its nose. It hovers about heavily, aimlessly; picks up in its mouth all what it finds (leaves, branches, filth, &c.), dropping it out again. If its way is barred with a hat, it is frightened, stops, but does not try to turn the obstacle. On finding a basin in the middle of an alley in the garden, it turns round about it excitedly, first in one sense, then in another, and so on. It starts impulsively to run, but gets entangled in the chain of another dog (amblyopic through occipital mutilation), and gets bitten. Frightened, it doubles itself up, without any attempt to escape from the attack. On being helped out by the attendant it begins to run about furiously, avoiding obstacles; but gets entangled and bitten again, and howls desperately until helped again. The animal, however, eats, drinks, and recognises its kennel.

(*b*) Chacma (Cynocephalus Porcarius). Adult intelligent female. Walks on all fours, or on the posterior limbs only, to command. Renders military salute, seizes and drops a stick, understanding words without gesture. Quick at new spontaneous adaptations; *e.g.*, raising and dropping the latch of a door through which she wants to get out. Distinguishes various foods, picking out without hesitation the preferred kinds. Observant and friendly; showing affection to another male cynocephalus recently operated, but without any erotic tendency even during the menstrual period, taking him in her arms, almost kissing him, pressing him to her breast with a satisfied and almost ecstatic look. This expression is still more evident in the case of two puppies, which she adopted from the first, and treats in a motherly way. These are the notes taken six days before the operation :—

May 23, 1892.—To-day a puppy accidentally got near her. She first looked at him with surprise and curiosity, then took him up gently on one arm, caressing him, and showing all the care and devotion which a mother shows to her child. She put him on the floor, followed him about as far as her chain would allow, gently picked it up and carried it back; then, fearing to lose it, she would keep it in her arms, or on her lap with one arm passed under the chest. Nothing induced her to let it go. If, when the puppy was on the floor, one called or whistled it to come, she understood the signal at once, picked it up and kept it close to her body. If one threatened her with a stick or otherwise, she became frantic, holding the puppy firmly but yet tenderly, and climbing to the top of the grating for safety. With great difficulty it was removed from her, but was restored soon after. She showed great pleasure, and as if grateful to me, came several times near me, holding out her hand for a caress.

A second puppy was placed near her, and began to prowl around. She examined, touched and sniffed it, but did not pick it up. But, when it began to move away, she seized it by the tail and pulled it to her; this was repeated several times. When she moved about she kept holding one of the puppies under one arm, and pulling the other along by the tail with the other hand.

It was impossible to get them away from her; and when anybody came near, she held both, each with one arm, and endeavoured to place them out of reach. In order to make her release them it was necessary to resort to a jet of water, of which she has the greatest horror, the very sight of the apparatus throwing her into the wildest excitement. Now, in spite of the

jet, she for a considerable time stuck to the two puppies, trying to shelter herself anyhow. At last she dropped the second puppy and climbed to the top of the grating with the other under her arm. Tired out at last, she gently deposited it on the ground and again climbed to the top.

May 29.—Operation on the left frontal lobe. About $3\frac{1}{2}$ grammes of cerebral tissue were removed, occupying over 2 sq. cm. in front of the foot of the first and second frontal convolution. Slight hæmorrhage; strict antiseptic precautions.

The next day she was feverish in the morning; but the temperature became normal in the afternoon. She was rather in a low state, but still could be fed.

June 2.—Right paresis. She uses the four limbs to walk, but is strong enough not to fall on the weakened side. In climbing, she uses the left limbs as chief points of support; the right hand, is stronger than the corresponding arm, which affects a paralytic posture during rest. Even with threats one does not elicit the military salute; she cannot pick up objects with the right hand, and when made to use it, she makes great efforts, and the object easily falls from the hand; she cannot carry it to her mouth. The posterior limbs are flexed; she cannot, as before, stand upright on her feet.

The external segment of the visual field is much enfeebled in the right eye; the nature of objects presented is not recognised therein, as it is in the internal segment. The experiment is decisive when a cherry is offered to her.

June 5.—Walks well; no "mouvement de manège." Does not use the left hand, only as when a cherry is seized or she tries to open a door, or raise herself to see what is on a table. She no longer gives the right hand, nor salute (*i.e.*, stands on the hind legs, seize the tail with the left hand, and carry the right to the forehead military-wise). The diapason is heard on both sides equally. The cherry is clearly perceived only on arriving from outwards to the middle line, or visual axis, of the right eye. She scratches the right side of the head and face with the right foot or left hand.

She is still susceptible of feelings and new adaptations. Her favourite puppy being offered, she takes it up, hugs it tenderly with the left arm only. She hunts for fleas with the teeth and left fingers; and resents any attempt to remove the dog. She cannot climb on a chair with the puppy; she places it on the chair first, and climbs afterwards. She did not care for, and avoided, the second puppy. Out of four she cares only for the

one she had previously taken to, and keeps it closely when they are called away.

June 26.—The paretic limbs have been recovering stength; for fine movements she still prefers the left hand; when compelled to use the right she is still uncertain. Vision is not yet perfect in the affected region.

August 16.—Second operation on the right frontal lobe. The trephine is applied to the point corresponding to the root of the frontal convolutions. Faradic excitations are followed by contractions of the left limbs, face, and peri-cranium respectively. Movements of the ear alone can be elicited by excitation of a point near that where movements of the peri-cranium and ear are obtained. About four grammes of frontal brain substance are removed. The incision falls accurately in front of the first and second frontal convolutions; and all the substance so separated is carefully removed. Much hæmorrhage. Two hours after the operation the animal drinks a glass of milk.

August 19.—Considerable swelling over the seat of the operation; no fever. The animal stands on its hind legs, but the left gives but uncertain support. On walking on all fours, the left arm drags. Tendency to rotation to the right.

Aimless instability of action. Reflexes exaggerated on both sides. Moderate excitations (by touching) produce generalised disorderly spasms. Left eye almost completely blind.

August 24.—Less rotation to the right. Still much instability; the animal climbs up and down a chair placed near, upsets it, pulls and shakes it about, opens and shuts a grating, pulls about her chain, and begins to rotate when not engaged in some of these actions. All this is done in a listless, automatic manner. She avoids impending falls or hurts, but immediately exposes herself again to the same accidents. She takes no interest in her neighbours and their actions, does not respond to calls, and manifests a quite unusual feeling of terror. She is even afraid of the other cynocephalus, whom she previously used to hug with evident pleasure. She has lost her previous interests, *e.g.*, in the puppy, of whom she is even afraid if placed near her.

Reflex hyperexcitability diminished. Slight hyperæsthesia on the left side. The right ear seems more sensitive to the diapason than the left. In order to test taste, two similar pieces, of sugar and plaster respectively, were offered; she took the one nearer to her—the plaster—chewed it, and swallowed it, without apparent feeling of pleasure or pain. The same happened with

the sugar, which she chewed and swallowed automatically. The experiment gave the same results a second time, when she again took the plaster without paying any of the minute attention to it as she used to do when any strange food was offered.

Vision was tested by different methods, and considerable impairment was constantly proved to exist. A fig, after being carried over a wide area of the external visual field of the left eye only, was placed close to its left hand, without the animal taking any notice of it; whilst it immediately made efforts to seize another fig placed at some distance on the right side. This absence of perception was noted in all meridians of the visual field of the left eye; the object was seen only on entering that of the right eye.

October 20.—The same automatism and semi-consciousness are still observed in all the psychical and motor manifestations of the animal. Rotation to right still considerable. On a loud call she shakes herself; and, after much practice, she attempts the military salute; and shows some feeling when caressed, and avidity when offered some food, but only for a short time together. The habitual state is still one of indifference; and she speedily relapses into an aimless, automatic mode of life, without any interest in her surroundings. Under novel conditions she is not capable of new adaptations, as she used to be. She no longer plays with her two companions (cynocephali), and remains indifferent, or pays but transient attention when they come near. She is not jealous when they are petted. Threatened, she evinces fear, but does not show any of the signs of self-defence and revolt she used to evince with so much energy. A feeling of indefinite and baseless terror is manifested, as soon as anyone goes near her, or her automatic agitation is suspended by a sudden call, or by any other new impression.

The movements of the left side are more precise, but not quite normal, though she uses it to take food, or steady herself in saluting. The vision of the left eye has, to a great extent, returned; but, though it is difficult to determine the degree with precision, it is certain that the internal portion of the visual field is more acute.

She continued much in the same state till January 21, 1893; and still has some tendency to rotation. She does not use much the left arm. She walks with agility and shows no trace of paralysis, using for that purpose the left arm. Sensibility intact in every sphere. She is killed with chloroform.

The left hemisphere looks larger than the right. The middle

and inferior frontal convolutions are destroyed as far as their insertion into the anterior Rolandic gyrus, which is almost intact. (fig. 1). The superior frontal has hardly been touched as far as its anterior extremity. In the right hemisphere, the superior frontal has been destroyed up to its root, excepting a small portion communicating with the rest of the brain by a narrow bridge (2 mm. in width). The second, or mid-frontal, is more extensively destroyed: of the third frontal, only the anterior and central portions are less destroyed (fig. 2).

(*c*) A young female cynocephalus, had been an inmate for about two months, whilst she was well treated and had become domesticated. She first menstruated during this period, and is much attached to one of the servants. She is docile, obedient, inquisitive and lively.

On *August* 2, 1893, I freely expose the right anterior hemisphere. Faradic excitation provokes no contraction in front of the points *a* and *b*. In *a*, movements of the trunk and pelvis were observed; in *b*, movements upwards and forwards of the ear, of dilatation of the palpebral slit, with elevation of the upper lid, and of dilatation of the pupil (chiefly on the same side). Repeated observations constantly gave the same results. Slight increase of current strength, or displacement of the electrodes 1 or 2 mm. backwards, movements of the hand (extension of the fingers chiefly) ensued. By a deep incision with a sharp curved blade I remove all the portion, one millimeter, in front of the points *a* and *b*. About six grammes of cerebral substance are removed. The wound was cleaned, under strict antiseptic precautions, and closed in the usual manner.

August 3.—Sleepy the whole day. Sits, does not lie down. No deviation of head and eyes. Uses the hands, preferably the right; walks about the cage normally. Drinks some milk.

August 4.—Same condition; is sick once.

August 7.—Has recovered from the operation; the wound has healed by first intention.

This animal is to all intents and purposes in a normal condition, save in two particulars, which only a searching examination can reveal. She is subject to fear, and has left external hemianopsia. She walks well; no rotary movements. Turns the head about with no difficulty to either side; is as rapid and agile as before. She perceives contact equally all over, including head and neck. Sudden prickings with a fine needle are followed with the most lively reactions.

If vision is prevented by means of sub-palpebral membranes of india-rubber (after cocainisation), the animal is found to have normal muscular sense. A peach brought near her hands is seized, bitten, skinned, and the pieces carried to the mouth with either hand, and with perfectly accurate movements. The sound of a diapason is heard, and causes fear, at the same distance of either ear. Smell and taste were not systematically tested.

From the day of the operation a defect of sight on the opposite side was noted. She was held by an attendant, whilst another kept her attention fixed on a point in front of her. From behind I bring a piece of peach to 10 or 15 cm. from the lateral or upper part of the visual field. When the object, travelling from outside towards the external angle of the right eye arrives in the field, it is immediately perceived, and sometimes rapidly seized and eaten. A similar piece brought towards the left eye has to be brought much farther forwards, almost as far as the median vertical plane, before it is perceived.

August 12.—On repeating the examination, the difference between the two eyes is found to be less precise and definite. She perceives something, but recognises it only when it reaches the left median vertical plane, the vision of the right eye being normal as before.

September 11.—She is still alert, agile, observant; she unties the string which closes the door, and pulls back the bolt and comes out. She is fond of tobacco. The end of a lighted cigar is thrown to her; she picks it up with left hand, burning herself; she speedily lets it go and licks her fingers; soon after she picks it up cautiously by the wet end, examines it, unrolls the leaves, which she chews one by one.

Up to January, 1894, no changes were observed, except that she was more irritable.

January 31, 1894.—Under morphia and chloroform, the anterior left hemisphere is exposed. The ascending frontal and the posterior portion of the first frontal convolution remain covered. Faradic excitation provokes contractions in the third frontal, where in front of the face centre (contractions in the inferior half of the face) a vertical rectangular area is determined, extending upwards to the second convolution, in which excitations arouse feeble opening of the lids, a slight raising of the eyebrow, and dilatation of the pupils, especially on the opposite side. At the origin of the first frontal, and a little in front, excitations provoke rotation of the head to the opposite side. No other movements could be elicited. (Repeated excitations of

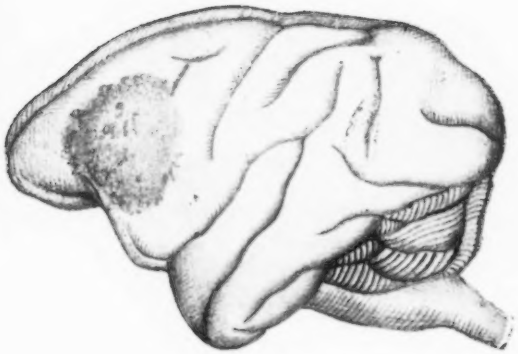


FIG. 1.

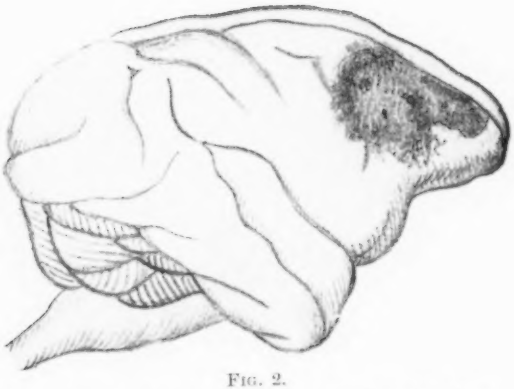


FIG. 2.

The Fissure of Rolando should be prolonged further down.

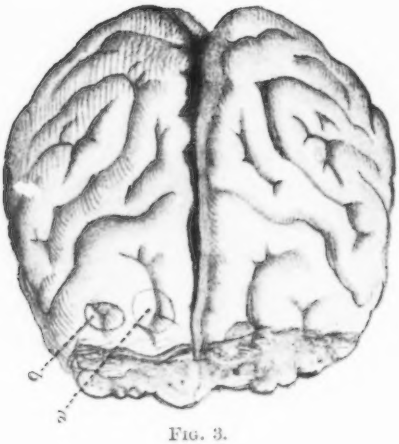


FIG. 3.

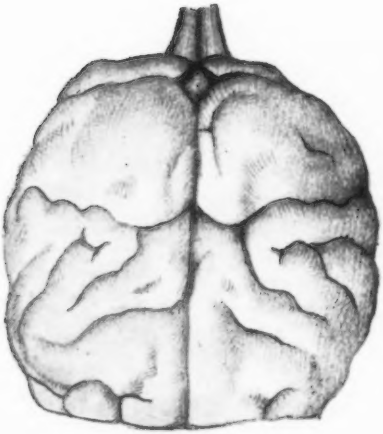


FIG. 4.

the pupillary centre produce persistent opening of the opposite lids, lasting about ten minutes.)

The incision is made immediately in front of the face centre, and behind that of the dilator iridis, and all the anterior portion of the brain (excepting the posterior third of the first frontal) is removed. Little hæmorrhage; antiseptic dressings. The animal remains for some hours in a somnolent condition; but opens the eyes when called. No vomiting nor fever.

February 2.—Remains in the cage with head bent, eyes half open, sleepy but sitting. When called she raises her head, slowly opens her eyes, looks about, and relapses into the same condition. When touched she wakes up, sometimes with a start or a cry, but does not try to escape nor looks alarmed. When let loose in a room, she walks about with measured steps. No deviation of the trunk nor paralysis, except in the right inferior half of the face.

February 9 and 14.—The notes of these two days may be taken together. Facial paresis not so marked, though evident. On walking, the lower part of the body looks arched. She can turn to either side, bend on herself, runs, without any trace of paralysis. Both sides of the body seem equal. Only she has lost her normal elasticity and suppleness; she is duller, heavier, more relaxed, even when urged on. She uses either hand, but preferably the left; there is still a certain precision but less decisiveness and security in the use of the right. No neck paralysis; all movements of the head, in every direction, are possible. She eats well, with avidity, swallows and utters cries as before. Tactile sensation preserved; she reacts to sudden contact of every portion of the body. Perhaps she feels less when the left ear is touched or pricked, as she does not move the part, or starts when it is done to the other ear. She scratches herself, and looks for parasites on both sides and with both hands. The muscular sense appears thus to be intact; no ataxia on walking, or performing delicate movements has been noticed.

As to sight, closure of the left eye does not interfere with walking, or running, all obstacles being accurately avoided. If, however, whilst she is at rest, a piece of sugar, tied to a string, is brought from the outside inwards, into the right visual field, it is perceived only when it is in a line with the visual axis. She then rapidly seizes it with the left hand and sometimes with the right, though it is less mobile. This experiment gave constant results. On the other hand, when the right eye is closed, the sugar is perceived and the movement for seizing it made the instant it is brought into the left field of vision. No marked difference be-

tween the upwards and downwards extent of the two fields of vision; the left internal field could not be examined on account of the fatigue.

Intellect.—Attitudes stupid, loss of physiognomical expression: apparently aimless walking. She runs incoherently from one end of the room to the other; when she reaches the wall she stops, returns to the opposite angle. Noticing a defect on the wall, where bits of plaster have become detached, she picks them up, chews and swallows them; the same with bits of filth on the ground. Pieces of plaster offered to her are taken and eaten as if they were sugar. It is not because she does not distinguish between these substances; for she takes longer to chew the plaster and seems at times to hesitate whether to go on or not; but she cannot resist the idea of sugar, which the appearance of the plaster has evoked. In all her actions there seems to be a defect of perception, which seems to be reduced to a more elementary level and lack some of the factors necessary to the formation of a more complete judgment. Her psychical life seems to be reduced to the existence of actual sensations.

Reflex excitability is exaggerated; every noise startles her, and she has lost her courage. On a threat she runs away, with incontinence of urine, or huddles herself up in a corner uttering lamentable cries. She displays no resource in her flight or attempts to hide herself, does not turn on the assailant; experience does not profit her, and as often as a sham attack is made upon her, she goes through the same actions as if she did not remember what had happened just before.

Formerly she used to cling affectionately to the legs of the attendant, caressed him, and nestled on him with the expression of the utmost pleasure and love. Now all seems indifferent; she stops at nothing; looks at everything but without fixing herself to anything. Her senses are excited but not attracted. She remains in this condition for months.

On *May* 4 she was chloroformed. The extent of the lesion is shown in fig. 3. No damage to the olfactory apparatus.

(*d*) Cynocephalus, female, good size, agile and cheerful. On close examination no defect of the senses, organs of motion, or of any other kind was discovered. She is inquisitive, cunning, acute, attached to her keepers. During the menstrual periods, which are regular, she is more excited and mobile than usual.

May 20, 1893.—Under morphia and chloroform she was trephined over both frontal lobes, one cm. in front of the Rolandic

sulci. Little hæmorrhage. The brain was exposed and two lines marked symmetrically on each side, passing in front of the roots of the frontal convolutions nearly corresponding to the præfrontal sulcus. Faradic excitations in front of these lines provokes no contractions, either in deep chloroformisation, or in mere morphinic somnolence. The whole of the brain substance in front of the lines was removed, and the wound dressed, under strict antiseptic precautions, in the usual manner.

May 21.—Stupor; marked reflexes to the least noise. She dips her snout in milk, hardly drinking any. Internal temperature 38·6 C. The next day the symptoms remained much the same. Appetite better. She was kept in her bed, and a clyster given.

May 27.—More awake, but the whole head is œdematous. She can, however, be examined. Sensation to pain and touch exaggerated, especially on the left side. The least contact, even to the tips of the hairs, is followed by reflex movements of the whole body, and turning of the head and eyes to the excited side. The slightest pinch, or pin-prick, elicits loud cries. A diapason suddenly set vibrating on either side astonishes her; she moves the head as if to avoid the strange noise. If the hand or any other object is brought towards the eyes, from whatever point in the visual field, she blinks and withdraws the head in fear. If a cherry, of which she is fond, is thrown, she fixes her eyes on it. If, suspended to a string, it is brought from above, or from the outside, into the visual field, she recognises it, and tries to take it, her movements being appropriate and co-ordinated with the mouth or the left hand. She walks badly, swaying, with head and body bent; the left leg drags, and the right arm little used. She hardly ever leaves her bed, nor takes any interest in things; but starts, emitting cries as if afraid or angry, to any noise, contact, or whenever she is attended to.

June 3.—The swelling has gone down. There is a local suppuration, and a superficial abscess has to be opened, cleaned and dressed. Her condition remains much the same; but she eats more, resists better, and walks, though with some uncertainty. The right leg is much paralysed.

June 7.—Suppuration has stopped. Wound granulates, and is still dressed antiseptically. No fever since the operation. The animal astonishes us all by its novel behaviour. She remains in the position given to her. No curiosity nor interest in what goes on. She spends hours sitting with head bent, as in a lump, often asleep. If a noise is made she shakes herself up, often with

a cry, raises the head, looks about, and relapses in her previous condition. The same occurs three or four times together if the same noise is repeated, until she is used to it. The only thing that excites her interest and makes her move is the sight of some favourite fruit dropped at a short distance. She then rises and runs to seize it. But if it is dropped further off she does not move; and it is difficult to say if it is because she does not see it, or through apathy. She occasionally scratches herself, but no longer hunts for fleas with her former rapidity and precision of movement. She resists if pushed away, and if she can cling to the place she occupies, she sticks to it. If pushed along by the nape of the neck she walks along semi-automatically, preferably along a wall, and stops in a corner, where she remains for a long time. When called aloud she looks up, but soon the head falls again, with frequent escape of saliva. She is very dirty, and does not move when passing her urine and fæces in which she sits. Placed with another menstruating female cynocephalus she stopped automatically, quite insensible to the erotic and lascivious manifestations of which she was the object, with no sign of sexual or racial interest whatever. And yet a noise startles and frightens her; a threat with a stick terrorises her, whilst her companion evidently recognises the sham nature of the demonstration. Fruits, sugar, and the like excite her more, but yet she lacks discrimination. A cherry into which a solution of quinine has been introduced is given to each monkey. The normal animal, after a bite or two, spits it out with a disappointed and irritated look, cleans her tongue with her hand, and crushes the deceptive fruit on the ground until no trace of it is left. The other chews, but stops after a while, salivating abundantly, and after some hesitation ends in swallowing it. After giving to each some good fruit, I give to each a cherry, dipped in the bitter solution. The first animal, as soon as it has tasted it, spits it out in anger; but soon after, seeing that the fruit is whole, picks it up again, examines it attentively, opens it carefully, eats the flesh and throws away the skin. The second goes through the same process of chewing, stopping, and finally swallowing the fruit.

A piece of sugar was given to the normal monkey; next a piece of plaster of the same shape and size and appearance. The animal picks it up, looks at it, sniffs it, and throws it away without tasting it. After giving it some natural cherries, I presented to it one made of wax. The monkey likewise scrutinizes it thoroughly, drops it as if to test the sound, tries to rub it, and

finally leaves it alone. The operated animal in both experiments displays a striking want of discrimination; it eats and swallows the plaster as it had done the sugar, and without any examination tries to chew a piece of wax, though not of the same shape as a cherry, giving us trouble when we tried to remove it from its mouth.

July 15.—This monkey has been kept under observation with reference to the changes in its temper, character, instincts and intellect. Her behaviour is altered, her physiognomy stupid, less mobile; the expression of the eyes is as if uncertain and cruel, devoid of any flashes of intelligence, curiosity, or sociability. Shows terror, even by shrieks and gnashing of teeth, when threatened or hurt, but never reacts aggressively. She is in a state of unrest; when placed in a large closed room, it walks aimlessly round it, always in the same direction, without stopping near any object or person. Any action done with apparent purpose remains incomplete, unfinished; if she runs towards a door she stops near it, goes back, runs to the door again, and so on several times.

She shows no affection nor gratefulness to people whom she previously loaded with caresses, as these animals are wont to do; she does not seem to know them, though they are ever ready to attend to her wants. Whenever approached for a caress she shows fear. When the attendant brings her food or fruit, she comes near and violently seizes the object of her avidity. None of her former friends can now caress her any longer. She is unsociable with the other monkeys, does not play; cannot overcome the least difficulties in her way by new adaptations, nor learn anything new, nor recover what she has forgotten. She picks up and takes to her mouth whatever she comes across. She is, however, somewhat cleaner.

The sexual instinct seems to be present; but the periods are less regular and abundant. She occasionally manifests impulses of cruelty quite foreign to her kind. One day whilst she was menstruating, another female cynocephalus came near her. She at once manifested her desire; but finding the other unable to satisfy it, she attacked her companion so furiously that she would have killed it if the keeper, armed with a stick, had not at once interfered.

A new psychomotor "tic" has recently been observed; every ten or fifteen minutes whilst walking, she stops suddenly, and placing the foot on the nape of the neck, bites her right buttock (sometimes the left also). Occasionally she proceeds to bite also

one heel, then the other, after which she quietly resumes her walk. No apparent local cause can be discovered for these actions, which are always identical, almost rhythmical. It is possible that at the outset some abnormal sensation has given a start to them, and that they afterwards were persevered in, governed by an uncontrollable automatism.

August 15.—Psychical state unchanged. Her movements have a stereotyped character, but are faultless. New adaptations remain impossible. She walks about, sits, looks for parasites all over the body, remains indifferent to everything but to the sight of her food. She often puts the parasites into her mouth, which she used not to do before. She also eats dry leaves or flowers. She no longer expresses her desire, at the time of menstruation, as she used to, by appropriate sounds and gestures.

No sensory defects are noticeable, on the closest investigation. But discrimination, and the higher co-ordinations of sensory factors on which the more complex psychical manifestations depend, and which are necessary to the preservation of the individual and the species, are reduced to mere rudiments. She now differentiates between a sugar plum and a piece of plaster. She eats the first with avidity; the second she breaks in pieces with her teeth, but spits them out on the floor. But then she takes one up to her mouth, throws it down again, and so on several times, at last swallowing it with avidity; she next takes a second, and so continues until the whole number has been disposed of. The whiteness of these fragments and their resemblance to those of a sugar plum have prevailed over her sense of taste. Her rudimentary inhibitory power has been overcome by the visual image; the other sensory factors did not intervene in the act of identification, in the judgment of analogy. She has crude sensations, but no co-ordination of simple presentations, into representations of higher complexity. Another monkey takes the plaster plum, examines and smells it, and either rejects it at once, or, if deluded by the visual resemblance into chewing it, spits it out and ignores the fragments; its comparative judgment and memory are certain, and spare it the struggle which, in the abnormal subject, was followed by a victory of the more elementary sensation, and by an irrational impulsive action.

August 23.—No change. The animal is chloroformed, and the right hemisphere exposed. Electrical examination of the cortex proved the existence of all the normal motor centres of the limbs and face. The brain is shown in fig. 4.

Conclusions.

Granted the objective accuracy of the facts just related, it will not be difficult to give a plausible explanation of the phenomena observed after ablation of the frontal lobes. But even if such a hypothesis be not universally accepted, the value of the observed facts will not be thereby diminished.

It is useless to discuss at great length Munk's views on this subject. Paralysis of the trunk-muscles does not always occur, especially when the incision falls accurately on the frontal lobes: and is in any case but temporary. Moreover, what connection can there be between such a paralysis and alterations of temper? It is strange that the Berlin physiologist should assign to such a function a centre so vast as to constitute a true province, far wider than that of the arm with its prodigious scope for movements of the highest complexity. And it is surprising to find the paralysis disappear some weeks or months after the experiment performed to disprove the narrow limits ascribed to the cortical area of a so much higher function.

We readily understand Munk, when with Meynert, Ferrier, and Goltz he refuses to admit a centre for a so-called intelligence, as he expresses himself. But it is astonishing that such an acute observer should have overlooked so many psychical symptoms, whilst attributing to the extensive frontal lobes, a function shown by Schäfer and Horsley to be localised in a much more modest region (internal aspect of marginal gyrus).

No scientific man at the present time dreams of galvanising into life the dead body of Gall's organology. Every one of the several sensori-motor areas of the cortex combines with the others in the formation of the psychical personality, whatever its characteristics may be; nay, the whole of the nervous system takes part in it. Nobody will contest Munk's thesis in this particular; but these remain to account for all the phenomena observed after the destruction of the frontal lobes, and which are not explained by the occurrence of a temporary trunk-muscle paralysis.

Neither does Ferrier's hypothesis satisfy us. Our facts point to more than a simple defect of attention correlated as much with a paresis of the ocular and cervical muscles as with a non-existing cause. Dogs and monkeys, a few weeks after the operation move their heads and eyes in every direction; and there remains nothing of this paralysis to explain the deep psychical changes observed by us. Such disorders, therefore, depend upon the absence of the frontal lobes, not as the motor centres of the head and neck, but as the anatomical substratum of special functions on which the formation and manifestation of psychical personality depend. The frontal lobes are not centres of inhibition. We speak too assuredly and too lightly of attention, and of inhibition, and one may feel surprised to find Ferrier identifying certain muscular co-ordinations with the very essence of attention, being thus led to spread over the whole frontal area the motor centres of the head and neck, in order to assign to it the functions of cortical seat of attention.

But we may ask ourselves whether there exists really any centre the function of which is to inhibit, hence whether there exists a faculty of attention. I reply distinctly, No.

Without attempting to go fully into the theories concerning inhibition and its centres, it will be granted that inhibition depends upon a general psycho-physiological process, and that every part of the nervous system becomes, under different circumstances, either an inhibitory or an inhibited centre. I upheld this view in 1885 in an essay on the act of walking, in which I modified in a more physiological sense the theory of Lauder Brunton, who explained inhibition by the law of interference. Taken in its strict sense, interference bears no likeness to inhibition: we must rather speak of an afflux of nerve-waves into a region excited by some definite stimulus or psychical representation, which afflux weakens the aptitude of other regions to fulfil its function. This mechanism can be illustrated by the action of any of the various centres, auditory (as when we are absorbed in listening to a musical performance), visual (as in presence of a great picture or object of scientific

interest), or motor (as in the case of a juggler or acrobat engaged in wonderful muscular display). Each respective cortical area becomes in its turn an inhibiting centre for the others. Physiology and psychology swarm with facts showing the error of those who assume a special cortical centre for inhibition.

We know how, when the mind is busy with a scientific problem, one becomes, as it were, deaf and blind. But if the auditory or visual centres are then strongly excited by a voice or an image, and a perception aroused, the flow of ideas is arrested or weakened as long as the psychical disturbance persists in another portion of the brain.

Thought, even when at work in the domain of pure conceptions, has in most men an inhibitory power. This is due to the fact that abstractions are resultants of very numerous psychical elementary factors, and determine the same dynamo-molecular processes in the innumerable nerve elements which co-operate in the formation of each concept. Hence the frontal lobes are no more centres of inhibition or attention than they are motor centres for the trunk, or head and neck muscles.

Overlooking the possible contention of some objectors, that the serious mental disturbances observed in the mutilated animals, depended on interference with the senses of taste and smell, my hypothesis is that the frontal lobes are the seat of co-ordination and fusion of the incoming and outgoing products of the several sensory and motor areas of the cortex. As the nervous waves from peripheral organs of reception (retinal rods, tactile organs, &c.) are transmitted from neurons of the first order to neurons of the second (mesocephalon, thalamus), and from these again to neurons of the third order (cortex), thus we may suppose that from the last-mentioned, nerve impulses travel to the frontal neurons of the highest order. The frontal lobes would thus sum up into series the products of the sensori-motor regions, as well as the emotive states which accompany all the perceptions, the fusion of which constitutes what has been called the *psychical tone* of the individual. Removal of the frontal

lobes does not so much interfere with the perceptions taken singly, as it does disaggregate the personality, and incapacitate for serialising and synthesizing groups of representations. The actual impressions, which serve to revive these groups, thus succeed one another disconnectedly under the influence of fortuitous external stimuli, and disappear without giving rise to associational processes in varied and recurrent succession. With the organ for the physiological fusion which forms the basis of association, disappear also the physical conditions underlying reminiscence, judgment, and discrimination, as is well shown in mutilated animals. Their agitation and motor incoherence depend upon the reflection of nerve impulses set up by stimuli through small sensori-motor arcs, without the intervention of the previously accumulated psychical co-efficients.

Fear is an immediate result of psychical disaggregation, from defective sense of personality, and unbalanced perception and judgment. As the oyster closes its valves on the approach of a cloud, thus the operated monkey trembles at the simulated hostility of its keeper, ceases to read on his face the smile of kindness, does not improve by past experience, nor perceive the means of escape at hand. Courage rests upon the treble basis of self-conscious force, rapid perception of the enemy's powers for offence or defence in relation to one's own, and the influence of certain feelings; our animals show an absence of all these characteristics. (We must not mistake for courage the impulsivity of certain animals; or men, insane or epileptic.) On the one hand, their effective nature, friendliness and sociability is impaired; on the other, their avidity becomes reckless and insatiable. From cleanly they become dirty; and their sexual functions are perturbed: the females menstruate irregularly; the males become, if not incompetent, at least incapable. All these symptoms depend not upon motor deficiences in the head, neck, or trunk muscles, but upon a dissolution of the psychical personality.

But even should a more acceptable hypothesis to explain the facts observed be hereafter framed, I feel at any rate certain of the accuracy of the observations themselves.

DEFECTIVE DEVELOPMENT OF THE CEREBELLUM IN A PUPPY.

BY J. S. RISIEN RUSSELL, M.D., M.R.C.P.

Research Scholar, British Medical Association, and Assistant Physician and Pathologist to the Metropolitan Hospital.

(From the Pathological Laboratory of University College, London.)

CONSIDERABLE interest is attached to the condition of the cerebellum in this puppy, both on its own merits, as illustrating some important points in the pathology of cerebellar disease, and also when considered in conjunction with a case of defective development of the cerebellum in a young cat, which I published in a recent number of this journal.[1]

I am indebted to Professor Victor Horsley for placing the animal at my disposal; and my best thanks are also due to him for allowing me to make the necessary examination of its central nervous system in the Pathological Laboratory of University College.

The animal in question is one of a litter of puppies all similarly affected, though born of healthy parents, both of whom were prize dogs. Unfortunately this was the only puppy obtained for examination, and came into Professor Horsley's possession through the kindness of Mr. Sewell, F.R.C.V.S.

During life it presented in a marked degree the clinical picture which we are in the habit of associating with some

[1] BRAIN, part i., 1895.

defect or disease of the cerebellum. Inco-ordination[1] was so great that in attempting to walk the animal fell, sometimes in one direction, sometimes in another, there being no one direction in which it was inclined to fall more frequently than in any other. When standing, and even when sitting there was almost constant oscillation of the head and trunk. The most careful examination failed to detect any clinical symptom that might be taken as an indication that one lateral half of the cerebellum was more affected than the other, and the correctness of this was borne out by the subsequent *post-mortem* examination of the central nervous system.

The animal was killed by an overdose of chloroform, and the central nervous system placed immediately in Müller's fluid for preservation. Sections were subsequently cut in celloidin, stained by Schäfer's modification of Pal's method, picro-carmine, aniline blue-black, hæmatoxylin, eosin, &c., and examined microscopically.

In the present description of the results of the macroscopic and microscopic examination of the central nervous system, the condition of the cerebellum will be dealt with first, those structures in other parts of the central nervous system which are known to be intimately connected with the cerebellum will be next considered, and finally the deductions which seem warranted will be submitted.

The Cerebellum.

On macroscopic examination of the organ it was evident that it was reduced in size so as to be about three-fourths the size of that of a normal puppy; but it was quite symmetrical, and the general contour of the organ was well preserved. Its convolutions and sulci appeared to be as much differentiated as in the normal cerebellum, and but for its general reduction in size, and the fact that the breadth

[1] I think it well to mention in this connection that I am familiar with the fact that the co-ordination of a normal puppy is not of the best, and that I am in a position to state that the inco-ordination presented by this animal was far in excess of anything I have ever seen in the normal puppy, and left no doubt in the minds of all of us who saw it that some defect of the cerebellum must be the cause of this.

of the convolutions was diminished, it might have passed as such.

Sections through the organ showed the proportion of grey and white matter to be on the whole not much altered in some places, the general normal arrangement being also preserved, but the folia looking somewhat skrunken. In other parts the cortex of the cerebellum was distinctly of less depth than it ought to have been, while in other places, and these were fewer, it appeared to be of greater depth than the normal.

The microscopic examination of the organ confirmed the general impression obtained from the macroscopic examination, inasmuch as in parts the proportion of grey cerebellar cortex to white matter was, as nearly as could be judged, normal, while at other parts the cortex was distinctly reduced in thickness, and in a few places it was of greater thickness than it ought to have been. Further examination revealed the important fact that the different layers of the cortex of the cerebellum were variously altered in their relation to each other. Thus in one part the molecular layer was three or more times the proper depth as compared with the normal or with the granular layer at this part (see fig. 1); while in another place the molecular layer was about one third or a half the depth of the normal, or as compared with what it ought to have been to be in proportion with the granular layer at the same point (see fig. 2). In other parts again the depth of the molecular layer, as compared with the normal or as compared with the granular layer, was as nearly as possible normal. In some parts of the cortex the molecular layer was of much greater depth than normal at one point, and greatly reduced in thickness at another, the transition from the one to the other being quite abrupt. A good idea of the extremes which were met with in places, side by side, may be obtained by comparing fig. 1, in which the molecular layer is greatly increased in depth, with fig. 2, in which the same layer is greatly reduced in depth. Some idea of the abruptness of the transition from the one condition to the other may be obtained from an examination of fig. 2, as in the extreme left of the

figure, beyond the cluster of cells of Purkinje, the molecular layer may be seen to be about four times the depth of that part of it above the cluster of cells and throughout the rest of the upper margin of the specimen.[1]

All that has been said with regard to the variations in the depth of the molecular layer in different parts, applies also to the granular layer, which presented all variations from the normal, being increased in thickness in some places, reduced in others, and preserving a fairly normal thickness in others.

Striking as were the appearances presented by the molecular and granular layers of the cortex of the cerebellum, the condition and arrangement of the cells of Purkinje were of still greater interest. In large parts of the cerebellum these cells were completely absent (see fig. 3), in other parts one or two were to be seen here and there, while in a few places irregular groups or clusters of these cells were to be seen, the outer margin of the cluster being at the junction of the molecular and granular layers, while the cells invaded the granular layer to a considerable extent (see figs. 1 and 2). One or other of the conditions already described was that met with as a rule, but in some places there was a greater tendency to regular arrangement of the cells of Purkinje in a single line in their normal position between the molecular and granular layers: but even here the distances between the cells were very irregular and often much greater than normal (see fig. 4).

Not only the arrangement of the cells of Purkinje was altered, they also presented all degrees of variation in shape and size. While in places these cells varied no more in shape and size than in the normal cerebellum, in other parts which formed the greater proportion, the cells presented the most varied shapes and sizes, many being much below the normal size.

One other point to be noted is the fact that the greatest paucity of the cells of Purkinje was met with in the lateral

[1] The photograph does not show this point as well as it might have done had a little more of the molecular layer to the left of the cluster of cells of Purkinje been included in the field.

lobes of the cerebellum. In the middle lobe, on the other hand, there was more regularity of arrangement, and a greater richness in these cells.

Corpus Dentatum.

This structure was absolutely intact on both sides, as seen in stained specimens both on macroscopic and microscopic examination. Its cells were normally arranged and individually presented no abnormal appearances.

Cerebellar Peduncles.

These were all reduced in size in proportion to the general reduction in the size of the cerebellum, but none of them showed any evidence of sclerosis or recent degeneration.

The Medulla Oblongata.

Inferior Olives.—The structures to which attention was primarily directed in this part of the central nervous system was naturally the inferior olives, owing to the intimate association known to exist between these structures and the cerebellum. They were of perfectly normal macroscopic appearance, and under the microscope no evidence of any abnormality could be detected, either in the characters of their component cells, or in their arrangement (see fig. 5).

The Restiform Bodies.—The region occupied by these peduncles of the cerebellum on both sides showed no evident defect in the way of absence, sclerosis, or more recent degeneration. Nor was any sclerosis or degeneration to be traced to either inferior olive.

The Spinal Cord.

Nothing abnormal could be detected in this structure. None of the tracts known, or supposed to be connected with the cerebellum, showed any evident reduction in size, sclerosis or recent degeneration. The grey matter was everywhere normal in appearance.

The Red Nucleus.

Inasmuch as experimental evidence has proved that so many of the fibres which leave the cerebellum by its superior peduncles, and which degenerate after ablation of some part of the organ, terminate in the red nucleus, careful search was made for any sign of defect in this structure, with negative result.

The Optic Thalamus.

Like the red nucleus, the optic thalamus is the recipient of a large number of the fibres which leave the cerebellum by its superior peduncles, as has been demonstrated by the degeneration method after ablation of parts of the cerebellum, so that special attention was paid to the appearances presented by this structure, but nothing abnormal was found.

The Cerebral Cortex.

To the unaided eye the appearances seemed quite normal; there was no detectable atrophy or shrinking of the convolutions, especial attention being paid to the condition of those of the sigmoid and adjacent gyri.

Auditory Nerve and Labyrinth.

No disease or defect was found in connection with these structures to account for the loss of power of equilibration which was so prominent a symptom during life.

Concluding Remarks.

The appearances met with in the cerebellum in this animal made it clear that the defects were of congenital origin, as indeed must be clear from the description of them contained in this paper. There was nothing in the appearances to suggest the existence of any disease of a progressive character, it is therefore interesting to find so much disturbance of equilibration, for animals deprived of even the half of the cerebellum, experimentally, regain their powers of

equilibration in a remarkably short time. Possibly the absence of compensation in this puppy was due to the fact that those structures by which compensation is ordinarily brought about, after lesions of the cerebellum, were themselves in an imperfect state of development at so early an age.

The next point which calls for comment is the fact that a defect limited to the cortex of the cerebellum, and affecting in such a great measure the cells of Purkinje, was attended during the animal's life, with symptoms which led those who saw the puppy to suspect some defect of the cerebellum.[1]

This in itself is a fact of no small importance: but as important is the fact that in spite of these changes in the cortex of the cerebellum, no evidence of any defect in the inferior olivary bodies could be detected. That no changes could be detected in the inferior olives is not surprising when we remember that the corpus dentatum was, as far as could be judged, absolutely intact on both sides. The intimate relationship which exists between the corpus dentatum and inferior olive has been too abundantly proved by experimental and other evidence to need further emphasis here: but it is gratifying to find confirmatory evidence of this fact in the animal under consideration.

The contrast between the condition of things in this puppy and that which existed in the cat,[2] to which I have already alluded, is so striking, that I cannot refrain from calling attention to it. Whereas in this puppy the defect was limited to the cortex of the cerebellum, and with the corpus dentatum intact on both sides, the inferior olives were also both intact, in the cat the defect (a unilateral one) involved the corpus dentatum to such an extent on the affected side that its only representatives were a few scattered cells in the region where it should have been found, and in conjunction with this condition the opposite inferior olive was extremely diminutive, contrasting markedly with its fellow. More important evidence, short of experimental, of the all-

[1] *Cf.* Herringham and Andrewes, *St. Bartholomew's Hospital Reports*, vol. xxiv.

[2] *Loc. cit.*

important relationship which exists between the corpus dentatum and inferior olive, as contrasted with any relationship between the cells of Purkinje and the inferior olive, could not easily be obtained.

DESCRIPTION OF PLATE.

FIG. 1.—Showing increased depth of the molecular layer of the cerebellum and a large cluster of cells of Purkinje to the right of the figure and an absence of these cells to the left of it.

FIG. 2.—Marked diminution in the depth of the molecular layer throughout the greater part of the figure, but greatly increased depth of it to the extreme left of it. Scarcity of cells of Purkinje except at the point of transition from the increased depth to the diminished depth of the molecular layer, where there is a group of them.

FIG. 3.—Complete absence of the cells of Purkinje, and diminution in the depth of the molecular layer.

FIG. 4.—Cells of Purkinje arranged in single line between the molecular and granular layers, but at very irregular distances from each other.

FIG. 5.—The inferior olivary bodies presenting no abnormal appearances

Fig. 1

Fig. 2.

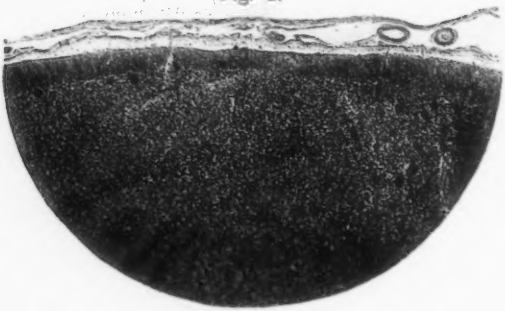


Fig. 3.

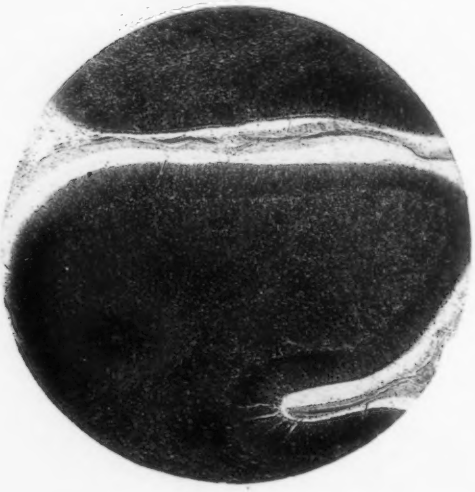


Fig. 4.

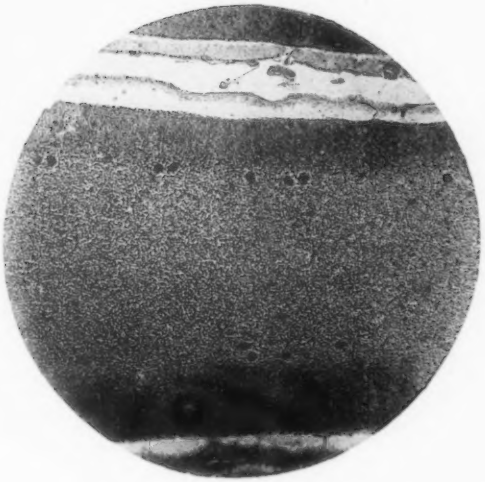
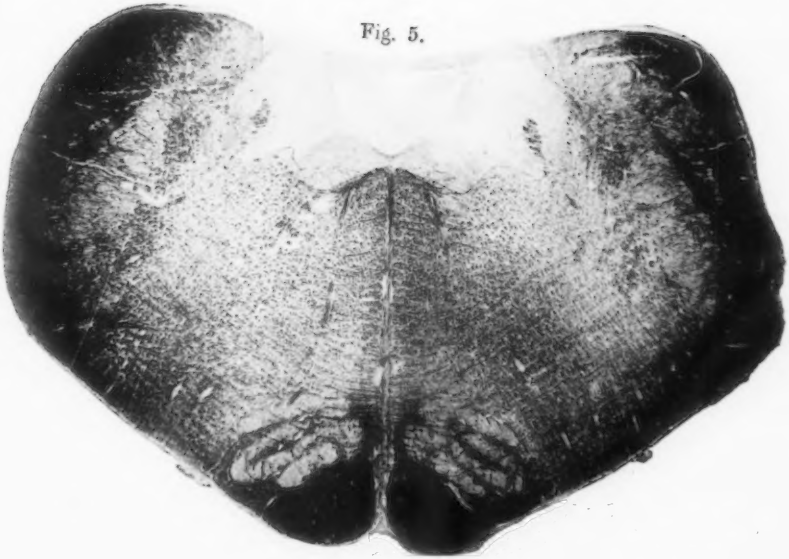


Fig. 5.



Danielsson & Co., London. Collotype.

DR. RISIEN RUSSELL ON THE DEFECTIVE DEVELOPMENT OF THE CEREBELLUM.

ON TUMOURS OF THE CORPUS CALLOSUM, WITH AN ACCOUNT OF A CASE.

BY W. B. RANSOM, M.A., M.D., M.R.C.P.
Physician to the General Hospital, Nottingham.

THE paper is based on the following case:—

Mrs. E. W., aged 24, married two and a half years, but never pregnant, came on September 8, 1893, as an out-patient to the General Hospital, Nottingham, for general nervousness and fits.

It appeared that her family were of an excitable, neurotic disposition, and that since marriage she and her husband had led a restless, pleasure-seeking life, and indulging rather freely in alcohol, although not so as to cause intoxication. There was no history of syphilis, congenital or acquired.

She was said never to have had any fits till two years ago, when the first one occurred after a mental shock. The fits have occurred as often as every other day, but for the last few months only once a week. The fit is preceded by a few moments of general excitement, then occurs a peculiar sensation in the temples; loss of consciousness and spasms following. These consist successively of twitching of the left hand and arm, left leg, left side of face, and deviation of the head and eyes to the left. The tongue has not been bitten, but urine may escape involuntarily.

The fits only last a few minutes, and are not followed by hysteroid attacks. The patient's natural excitability has much increased during the last year, and headaches, to which she has all her life been liable, have become more frequent and severe. The pain is most intense in the temples and the nape of the neck. For the last six months the eyes have become prominent and staring, but the neck has not enlarged, and she has not complained of palpitation or sweats. The pulse rate on this date = 120.

Under treatment with moderate doses of bromide the fits stopped entirely, but the mental excitability increased so that her family could hardly manage her.

On *October* 18 she was admitted as an in-patient, being then in a semi-maniacal condition. Her violence was at first such as only to be controlled by an injection of hyoscyamine, but began to lessen on the second day after admission.

State on October 20.—The patient is still restless and excitable and rambles in her talk, having some ideas of persecution. She says her headache has gone. The facial expression is anxious and frightened; there is marked exophthalmos, and the voice is somewhat tremulous. The temperature is normal; the pulse is very small and soft = 120; thyroid gland not enlarged. The heart and lungs are normal to examination. Though the tongue is coated, the appetite is good, and there is no sickness or other disturbance of the digestive system. The bowels are rather constipated. Generative organs normal. There is no paralysis, spasm, or alteration of general sensation, and the special senses appear normal.

There are slight general tremors of limbs on movement, and the gait is a little uncertain; she seems to totter towards the left. The knee-jerks and other reflexes are normal. The pupils are normal. There is distinct commencing optic neuritis in each eye.

Progress of Case, November 1.—Although occasionally noisy and excitable, the patient has on the whole improved, the attacks of restlessness soon yielding to bromide and chloral. The pupils have been somewhat dilated.

November 17.—Up till yesterday there was marked improvement, mental and physical, and sedatives have hardly been needed. [Since October 28 the patient has been taking pot. iodid. and liq. hydrarg. perchlor.] She has been free from headache, and has become fairly rational. Pulse varies, 70-100. Last night the temperature rose to 101·4°, but is 97° this morning. To-day she had a fit. When seen in it she appeared quite unconscious; the left side of the mouth was drawn up, the eyes were turned to the left, the left hand was clenched, but the arm rigidly extended, as was the left leg, which twitched occasionally. The tongue was not bitten, neither were the sphincters relaxed. The attack lasted about three minutes, and was followed by bi-temporal headache.

November 23.—Another similar fit occurred early this morning. When visited four hours later she said she felt "queer," "headachy," and as if about to have another fit. She then

appeared gradually to lose consciousness, and could not be roused for four minutes. There were no tonic spasms, though the left arm moved in a purposeless manner. The pupils were medium-sized and equal. Pulse = 82; respirations = 17. On the return of consciousness the patient vomited food, the pulse rose to 132, and the pupils became temporarily dilated. She was found to have double ankle clonus, and loss of tactile sensibility on both legs, but not on the arms or face.

November 28.—There have been three more fits, followed by sickness. Between the attacks the patient's mental condition is calm and rational, in fact nearly normal. The pupils are normal, the knee-jerks of normal vigour, the ankle clonus is no longer obtained and the anæsthesia of the legs has disappeared. There is a doubtful slight paresis of the left angle of the mouth. No deviation of the tongue. Right grip = 40; left grip = 35. She walks well and steadily.

There is well-marked double optic neuritis, most intense in the right eye, but the vision is good, as are the other special senses. The temperature has been normal.

December 8.—The mental and physical improvement has continued. The patient is quiet and intelligent, and though still obviously nervous, not more so than she has always been. She has gained 7lbs. in weight, is free from headache and sickness, and has had no more fits. The exophthalmos is less, though still present: P. varies from 90-120.

The optic neuritis is distinct, but the effusion is slightly paler.

December 9.—To-day the patient was sent home.

February 12, 1894.—During the two past months the patient has been seen occasionally, and on the whole the improvement seemed to be maintained, though domestic affairs at times brought out a latent irritability. Except, however, that the optic neuritis persisted there was neither sign nor symptom of serious organic disease of the brain.

It was, therefore, a surprise to hear that this morning she had suddenly died. I immediately went to her residence and though I could get no satisfactory information as to the exact mode of death, obtained permission to open the head. When this was done there was found between the cerebral hemispheres a tumour of the size of a Tangerine orange, springing from the middle of the corpus callosum and bulging slightly into each lateral ventricle. The growth was of a greyish colour and very soft. On microscopical examination it proved to be an oval-celled sarcoma. Its margins were well-defined, and the rest of the brain was healthy.

Commentary.

It will probably be conceded that an exact diagnosis of the position of this tumour during life was hardly possible. Indeed, the diagnosis of cerebral tumour at all, although inclined to by me, was very uncertain. The history of family and personal neurosis, of a somewhat dissipated life culminating in attacks of epilepsy, great mental excitement and temporary mania, suggested a generalised degenerative process in the cortex likely to lead to dementia or possibly general paralysis of the insane. The exophthalmos and rapid pulse further suggested a development of Graves' disease, which is so frequently met with among the inmates of asylums. The absence of vomiting (except after fits), the comparative slightness of headache, and the very marked mental and physical improvement which took place in hospital, all seemed to negative the view of the existence of such a coarse brain lesion as tumour. It was only the persistence of well-marked optic neuritis which caused one to look upon the improvement as but temporary and to bear in mind the probability of the recurrence of serious symptoms. Yet this fact alone could not justify an entirely unfavourable prognosis, for cases not a few have been recorded in which optic neuritis has been found without grave brain or kidney mischief, and which have made good recoveries. I have myself observed carefully the case of a young man who suffered from very severe vomiting, headache, and intense optic neuritis, who, in the course of his illness, had vertigo and epileptiform attacks, and in whom renal disease, syphilis, and plumbism could be almost certainly excluded, who yet made a complete recovery, and is now, two years later, apparently in perfect health. But such cases are rare. In young women "benign" optic neuritis is usually associated with severe anæmia, and the present patient showed no signs of anæmia or syphilis. I therefore felt it probable, when she went home apparently on the high road to health, that disease was still there and that before long we should see her again.

With regard to the relation of symptoms to the position of the growth, many points of interest arise.

I originally took the patient into hospital in order to try to localise the lesion causing the fits with a view to possible operation, and the details of the convulsions were therefore accurately noted. In the few fits seen by us in hospital the spasms were definitely one-sided, and in others, observed by her friends before admission, a distinct march of events was noticed—arm, leg, face—facts which suggested a sub-cortical lesion near the "motor" area and probably nearest to the arm centre. But the attacks after admission were very few, and there was an almost complete absence of disturbance of motor or sensory power in the affected limbs between the attacks. [Only for a day or two did there appear a very slight and doubtful weakening of the muscles on the left side of the face.] Such a diagnosis was therefore very doubtful, and the subsidence of the fits quite put aside the question of surgical intervention. The case becomes of considerable importance as a warning against hasty operation, showing, as it clearly does, that localised one-sided convulsions appearing in a definite order may be produced by a lesion remote from the "motor" area of the cortex or the fibres subjacent thereto. For the tumour, although it bulged somewhat into the lateral ventricles, did not infiltrate or perceptibly flatten either cortex or corona radiata. From its position, if it did irritate or destroy adjacent portions of the hemispheres, the parts most affected would have been the gyri fornicati or that part of the leg centre which comes over on to the mesial aspect, of which there was no clinical evidence.

The probable conclusion is that the epileptic spasms so closely resembling those usually caused by irritation of the cortical grey matter about the fissure of Rolando were in this case due to excitation of the fibres of the corpus callosum.

This view is strongly supported by the interesting experiments of Mott and Schäfer,[1] who found that electrical excitation of the longitudinally bisected corpus callosum produced localised movements on the side opposite to the

[1] Brain, part ii., 1890.

hemisphere with which the stimulated fibres are connected, the muscular contractions occurring as the electrodes were moved from before backwards successively in eye, head, arm, trunk and leg. They further found that if the grey matter of the connected Rolandic cortex were destroyed, these movements could no longer be obtained, although stimulation of the exposed corona radiata was still effective. From which facts they drew the probable inference that the callosal fibres were connected with the cells of the Rolandic area.

To the subject of the function of the fibres I shall return later. At present the clinical fact may be noted that their excitation may produce phenomena hardly, if at all distinguishable from those consequent on excitation of the "motor" area of the cortex—a further proof of the need of caution in advising operation in cerebral disease.

The symptoms in this case differed much from those described by Dr. Bristowe,[1] whose summary has been copied into text-books as diagnostic of tumours of the corpus callosum.

A comparison, however, of his cases with this and the few others of which I have found records, hardly seem to me to support his conclusions. Dr. Bristowe's two first characteristics—(1) The ingravescent character of the symptoms; (2) The gradual coming on of hemiplegia resembling that due to hæmorrhage or embolism—certainly do not distinguish a callosal growth from one in the white substance of the hemisphere or in the corpus striatum, and the same may be said of his two last points,—"the absence of implication of the oculo-motor nerves and of direct implication of other cerebral nerves, and lastly, death from coma." His fourth symptom—"the supervention of stupidity associated for the most part with extreme drowsiness, a puzzled, enquiring look when awake, a difficulty of getting food down the throat, and cessation of speech"—is certainly met with in other cases of brain tumour and was absent in my and in other cases of growth in the corpus callosum. Dr. Bris-

[1] Brain, October, 1884.

towe's third characteristic—"the association with the paralysis of one side, of vague hemiplegic symptoms of the other,"—when present is doubtless of value, but it may, as in my case, be totally absent. It is possible, however, that the double ankle clonus and partial anæsthesia of both legs, which occurred for a short time after one of the fits, was a point of some importance as indicating a lesion to some extent affecting both hemispheres.

Bruns[1] concludes that:—(1) When the symptoms of an organic brain lesion are present and these symptoms increase slowly and steadily as do those of a tumour; (2) when hemiparetic or paraparetic affections are superadded; (3) when well-marked dementia, to some extent contrasting with the slightness of the general tumour symptoms (headache, vomiting, convulsions, optic neuritis) is present; and (4) when no other symptoms exist which allow of another localisation, then a tumour of the corpus callosum is probable.

But it is obvious, and Bruns himself admits it, that tumours in other situations may produce the same combination of symptoms, and also that the symptoms of a growth in the corpus callosum may vary considerably according to the part involved.

I have described (*Lancet*, July 1, 1893) a case of tumour of the caudate nucleus in which the symptoms closely resembled the above combination.

Appended (p. 548) is a tabular analysis of eleven cases of tumour of the corpus callosum, from which some general conclusions may be drawn. From this table it will be seen that Bruns' diagnostic group of signs is but rarely well developed. Thus the evidence of tumour may be slight or absent.

Headache was absent in five of the eleven cases, and was well marked only in two.

Vomiting occurred only in one (excluding my case, in which it only occurred occasionally after a fit).

Optic neuritis was noted only in three and was stated to be absent in four.

[1] *Berl. Klin. Woch.*, 21, 1886.

The only sign of cerebral disease common to all (excepting one case of Glaeser,[1] of which the account is brief and imperfect, was *dementia*.

Dementia, in the form of monomania, was the only sign of brain disease in Berkley's[2] case, and in Oliver's[3] case visual hallucinations associated only with loss of colour vision and amblyopia suggested the diagnosis of hysteria; and in one of Brun's cases the dementia and paralytic symptoms were such as to lead Hitzig to diagnose general paralysis of the insane. In most cases it was the first symptom noted. Usually there was general failure of the intellectual powers, but in Berkley's case, except for his monomania, the patient was fairly acute. In all the slowly developed cases excitability was marked, maniacal outbreaks being common; but in my case the mental condition improved remarkably during the last two months of life.

Convulsions were present in two of the eleven cases (Glaeser, Ransom). In Glaeser's case there was left hemiplegia, with spasms of the paralysed limbs, but general epileptiform convulsions were the earliest symptoms of disease. In my case epileptic fits occurred as early as two years before death, in association with general excitability and occasional headache. The convulsions usually became general, but were more marked on the left side. It is of interest to note that in my case the head turned to the left, while in Glaeser's it turned to the right. [See Gotch and Parker, *British Medical Journal*, May 27, 1893, for the difference between men and monkeys in this respect.] My case further differed in that no paresis occurred in the convulsed parts, and the *post-mortem* examination showed no invasion of the motor path, which in Glaeser's case was apparently affected by the extensive growth.

Paralysis.—Some degree of paralysis was observed in seven of the eleven cases, and in six of these it took the form of hemiplegia, usually with some rigidity. In the hemiplegic cases the leg was earlier and more markedly affected

[1] *Berl. Klin. Woch.*, 1883, p. 809.
[2] *Amer. Journ. of Med. Sci.*, 1890, p. 578.
[3] Ref. in *Lancet*, August 22, 1891.

than the arm, and in one the face escaped. Aphasia was especially noted in one, and in another the speech was thick, like that of a general paralytic. In one of Dr. Bristowe's cases, left hemi-paresis was associated with some weakness of the opposite side.

In one case (Glaeser) there was paraplegia, the weakness and rigidity being more marked in one leg than the other. In this case, the notes of which are unfortunately meagre, the loss of power in the legs appears to have been the only symptom.

In all the cases with paralytic symptoms it was found that the tumour had invaded the white substance of the hemispheres, especially that opposite to the paralysed side. When there was no loss of motor power, either the tumour was small and did not infiltrate the centrum ovale, or it was situated at the anterior or hind end of the corpus callosum.

Sensation was less usually affected than motor power. In five of these cases it was found normal, and in two only was hemi-paresis associated with slight hemi-anæsthesia or hemi-analgesia. In the case with slight right hemi-anæsthesia (Bristowe) the growth in the anterior half of the corpus callosum extended some distance into the left centrum ovale; in that with slight right hemi-analgesia, the tumour was situated just in front of the splenium and extended slightly into the right precuneate lobule and rather more into the left central convolutions. The gyri fornicati were free, but the left optic thalamus was somewhat atrophied.

In one case only (Oliver) was the special senses markedly affected. Here vision became gradually lost in the left eye, while colour vision was impaired in the right; hearing was diminished in the left ear, and taste and smell were impaired. Visual hallucinations were the prominent feature.

The tumour in this case is described as having arisen from the knee of the corpus callosum, and pressed downwards on the optic nerves as well as on the uncinate gyri.

It would thus appear that the interference with vision was due to direct damage to the nerves, and not to injury of optic radiations or cortical visual centre.

The *reflexes* do not call for special comment, except that

there seems rather less tendency to increased tendon reflexes in the paretic limbs than might be expected with spastic hemiplegia. In only two cases were the knee-jerks found increased on the paralysed side, and the only occurrence of ankle clonus seems to have been what was found in my case just after a fit.

The pupil reflexes seem to have been normal, except in Oliver's case, of pressure on the optic nerves.

The *cranial nerves* almost invariably escaped; the only change noted being inequality of the pupils in three cases—including that of Oliver. But this inequality, as is well known, may be produced by pressure on the cortex, without involvement of the nerves at the base of the brain.

The *duration* of symptoms of disease varied from five weeks to three years.

Death occurred in five cases from gradual coma, usually associated with pyrexia; in two cases from multiple hæmorrhages, in two from pulmonary complications, and in two the end was sudden but of unexplained mechanism.

Diagnostic Conclusions.

From this analysis it appears doubtful whether a positive diagnosis of tumour of the corpus callosum can ever be made. The symptoms may closely resemble those not only of tumours of the central ganglia, centrum ovale, or frontal lobes, but also those of softening from thrombosis of the Sylvian artery and those of general paralysis of the insane and of other forms of insanity.

It appears that a tumour in this position can exist for long and attain considerable size without producing marked pressure symptoms. The tumour symptoms are generally slight. Paralysis may follow the invasion of a hemisphere by the growth, and usually takes the form of hemiplegia with rigidity, and is associated with little or no anæsthesia. The leg tends to be affected more than the arm, and the arm than the face. Occasionally the opposite limbs may share in the weakness, and then the diagnosis is greatly facilitated, as only cerebral softening, multiple tumours, or growths in the pons or medulla would be likely to produce the same combination.

Early and marked mental degeneration would appear to be a characteristic symptom. The cranial nerves are not affected. Convulsions may or may not be present, but are of little localising value. Vertigo and ataxy are absent.

To sum up, a tumour of the corpus callosum may be diagnosed as probable when there are present :—

(1) Gradual but marked mental change, in more acute cases taking the form of increasing stupor, in the more chronic appearing as insanity with delusions, irritability, and maniacal outbursts.

(2) Moderate development of tumour symptoms (headache, optic neuritis, vomiting).

(3) Hemiparesis with rigidity, with or without slight hemi-anæsthesia, and with or without convulsions.

If the opposite limbs are also slightly affected, the diagnosis is strengthened.

(4) Or convulsions without paresis—the spasms being bilateral, but more intense on one side.

(5) Integrity of the cranial nerves.

(6) Little or no change in the tendon reflexes.

This group of symptoms may, however, obviously be produced by other cerebral affections, and it is only when the paresis or spasms affect to some extent both sides, without other evidence of a multiple lesion, that any degree of certainty can be attained.

Relation of Symptoms to Function of Corpus Callosum.

Our knowledge of the anatomical and functional relations of the corpus callosum is at present so slight that but few conclusions can be drawn as to the dependence of the above described symptoms upon the lesion of that structure.

Koranyi[1] has shown that section of the corpus callosum of dogs produces no permanent paralysis or notable disturbance, though convulsions of the whole body may at times occur. I am not aware that these experiments have been repeated on monkeys. Several instances of its complete absence in man have been recorded, in which the

[1] *Pflüger's Archiv*, Bd. xlviii.

symptoms during life presented considerable variations. Thus Sir James Paget[1] found this body absent in a patient "not remarkable for any excellence or great defect of mind." Langdon Down,[2] in the brain of an idiot who could walk and run and say a few words, found absence of the corpus callosum, body of the fornix, and the soft commissure. Dunn[3] found no corpus callosum but an otherwise well-developed brain in the body of a man of whom there was no probability that he had been an idiot. In this case the body of the fornix was also absent.

Dr. Alexander Bruce[4] has given a full account of a case of his own, in which the corpus callosum was absent without marked mental defect, and has also made a careful digest of fifteen other cases of complete absence of this body, as well as others in which it was primarily defective and others in which its absence was apparently secondary to other cerebral lesions. The study of these cases leads to the conclusion that absence of the corpus callosum alone need not cause mental or bodily disorder, but that such are due to other associated brain defects. Dr. Bruce points out that the recorded facts lend no support to the anatomical and physiological doctrine of Meynert, that this structure co-ordinates corresponding convolutions in the two hemispheres, and that, in fact, this view is purely theoretical. So far as I know, the only experiments giving positive results on the function of the corpus callosum are those above referred to of Mott and Schäfer.[5] These clearly show that impulses may travel along the fibres of this body up to cells of the Rolandic convolutions, and excite these cells to liberate motor currents downwards. But they do not show with what structures these fibres are connected at the end distal from the Rolandic cells. Dr. Mott[6] has, however, in his report on this work to the British Medical Association, given a diagram in which this distal end terminates in a ramification over a corresponding

[1] *Med. Chi. Trans.*, vol. xxix. [2] *Med. Chi. Trans.*, vol. xliv.
[3] *Guy's Hospital Reports*, vol. xlvi., 1889. [4] BRAIN, July, 1889.
[5] *Ibid.*, 1890, p. 174. [6] *Brit. Med. Journal*, May 17, 1890.

"motor" cell in the opposite hemisphere—an arrangement which apparently depends merely on Meynert's theory.

If we seek information from the study of secondary degeneration, we find from the experiments of Sherrington[1] that lesions of the cortex, not only of the cord area, but of areas anterior, posterior and lateral to it, are followed by a degeneration of scattered fibres in the opposite hemisphere, their distribution not being further indicated. In Boyce's[2] experiments, after the removal of the whole of one cerebral hemisphere of a cat, degenerate fibres were traced across the corpus callosum into the other hemisphere which tend to turn *upwards*. Neither of these series of experiments teaches us the nutritive centre of the bulk of the callosal fibres, the normal direction of impulses along them, or their actual anatomical relations to the cortical cells. According to Mott's diagram, a callosal fibre is a process of a cortical cell in one hemisphere, ending in a dendron over a cortical cell in the other hemisphere; but the actual facts at our disposal give us no exact information as to one end of the fibre or the other. The comparatively small number and scattered distribution of fibres which degenerate after even extensive experimental lesions of the cortex, is in agreement with what is found in human brains after softening of the Rolandic cortex, where little or no degeneration has been traced across the corpus callosum, but is hardly compatible with the view that most of these fibres are processes of cortical cells.[3]

The alternative view, therefore, presents itself that some, at least, of the callosal fibres have their nutritive centre below the cortex. The case of tumour of the corpus callosum recorded by Berkley, appears to lend support to this

[1] *Journ. of Physiol.*, vol. x.

[2] *Brit. Med. Journ.*, Sept. 23, 1893.

[3] I have cut sections of the whole of the corpus callosum from a case of hemiplegia of two years' duration, in which there was marked softening of the left Rolandic area due to arterial thrombosis. Though there was well-marked descending degeneration in the left internal capsule and the crossed pyramidal tract of the cord, readily visible to the naked eye, in the corpus callosum one could only distinguish microscopically, in sections stained by Weigert's method, a few scattered degenerate fibres.

view. Here a small growth destroyed most of the fibres just behind the genu; the degeneration could be traced into the frontal lobes, and there was marked wasting of the frontal convolutions. One may conceive that, as in amyotrophic lateral sclerosis, the cells of the anterior cornu degenerate after destruction of the terminal ramifications of the pyramidal fibres, so the cells of the frontal cortex shrink after atrophy of fibres of the corpus callosum.

These considerations lead us to the views of Hamilton,[1] which have been expounded in a number of papers for several years, but which appear to have met with but little acceptance. Using the anatomical method, Hamilton arrived at the conclusion that the callosal fibres are not commissural for corresponding parts of the hemispheres, but that, starting from the cortex on one side they cross to the other hemisphere, arch down into the anterior limb of the inner capsule, and to some extent into the outer capsule, and end, for the most part, in the optic thalamus, in the cells of which they may possibly have their nutritive centre. The tracing of the path of fibres in the centrum ovale by the anatomical method alone is undoubtedly a task of great difficulty. It is quite possible, therefore, that, as Bruce suggests, some of Hamilton's arched "callosal" fibres may really descend from the gyrus fornicatus and not from the corpus callosum, but the evidence adduced by Hamilton appears too strong to be completely set aside. If callosal fibres have their trophic cells in the thalamus, then destruction of the corpus callosum need not lead to atrophy of the internal capsule, as has been argued against Hamilton by Bruce. The degenerations found in Berkley's case might, however, naturally follow. The most serious argument against the doctrine of Hamilton appears to be the statement (made by Bruce) that in the human infant up to three months the callosal system is non-medullated, while "almost the whole of both limbs of the internal capsule are medullated." Hamilton, however, speaks of the arching downwards of the corpus callosum as being very well seen in the

[1] *Proc. Roy. Soc.*, 1884; *Journ. of Anat. and Physiol.*, 1885; "Text-book of Pathology, 1894. [See also BRAIN, vol. viii. (1886), p. 145, and Beevor's criticism, *ibid.*, p. 377.]

four months human embryo. He also found the callosal arch naturally dissected out in a porencephalic brain. Finally, in the brains of certain lower mammalia, such as the armadillo, the corpus callosum has been figured by Rabl-Rückhard as turning downwards into the inner and outer capsules.

Hamilton's anatomical views, therefore, cannot be said to be overthrown, but on the contrary, have some amount of probability.

The stimulation experiments of Schäfer and Mott are in harmony with them. We may thus consider some of the fibres of the corpus callosum as a part of the afferent system, as conducting impulses upward, and those fibres which go to the Rolandic area may be looked upon as the afferent part of the kinæsthetic mechanism of cells and fibres. Those fibres which are connected with the frontal and occipital lobes, on the other hand, may conduct some of the stream of afferent impulses which excite perceptive and ideational cerebral processes. There are many grounds for considering the optic thalamus as one of the halting-places of sensory impulses. The specialised senses, such as touch and sight, are known not to travel by the anterior limb of the internal capsule on their way to the cortex, but their impulses form but a comparatively small proportion of the multitude that stream upward. The path by which currents from the cerebellum travel from the basal ganglia to the cortex, the paths of afferent impulses from the viscera and the muscles, and of the vague tactile sense which is often lost when the "motor" cortex is destroyed, may lie among these callosal fibres. The results of degeneration experiments make it further probable that these fibres do not actually join the cells of the cortex, but probably end in a ramification among the processes of those cells, as it is supposed do the pyramidal fibres of the cord with the protoplasmic processes of the cells of the anterior cornua.

Such a view of the structure and function of the corpus callosum is in harmony with the facts observed in cases of disease of that organ. A disordered sensorium is a prime condition of mental aberration; upon the regulated stream

of sub-conscious impressions depends the order of conscious processes. A tumour in the corpus callosum on this theory will now diminish, now increase to violence the force of this stream, and dementia or mania ensues. If the lesion irritate also those fibres ascending to the cells of the Rolandic or "cord" area of the cortex, violent action of those cells is produced and convulsions ensue. What happens when the growth destroys these fibres without involving the hemispheres as well is rather uncertain. Possibly some of the paresis observed may be thus caused; but Koranyi's experiments seem to show that in dogs, at least, the functional activity of the cells does not entirely depend on the integrity of these fibres, and in the reported cases of brain disease there has usually been too much involvement of the white substance of the hemispheres to allow of making deductions on this point. Experiments on monkeys are desirable to throw light on this point. One would expect to find after section of the corpus callosum in the monkey some paresis and affection of the muscular sense, which would probably be, however, only temporary. Experiments are also needed to study the degenerations consequent on destruction of the optic thalamus.

Pathological observations have so far thrown but little light on the relations of the optic thalamus to the cortex or to the corpus callosum. Though hyperæmia and small hæmorrhages have been often noticed in the thalami in cases of chorea, yet it is not certain that they are more abundant there than in other parts of the central nervous system. Hammond, in his original description of athetosis, attributed this disorder to a sclerosis of the corpus striatum and optic thalamus, and Charcot[1] found the posterior parts of the optic thalamus and corpus striatum affected in three cases of post-hemiplegic choreoid spasms; but the latter goes on to say that he has "often and often seen the optic thalamus and lenticular nucleus affected in their several parts with the most varied lesions and not the slightest trace of choreic movements resulting." In one case of

[1] *Lectures* (Syd. Soc., second series.)

"pre-hemiplegic chorea" Charcot found a small hæmorrhage in the posterior half of the optic thalamus.

Dr. Gowers[1] has recorded a case of right hemiplegia, recovered from in three months but followed by ataxy of the right arm, in which the only brain lesion found was a cicatrix in the left optic thalamus.

In this case, as in the last quoted one of Charcot, it appears probable that the lesion in the thalamus disturbed the adjacent motor fibres in the internal capsule. If the symptoms were produced by disturbance of *afferent* impulses, these cases indicate that early thalamus is chiefly connected with the cortex of its own side of the brain, and not, as it should be on Hamilton's theory, with the opposite hemisphere. At any rate the very interesting case of Dr. Gowers lends no support to Prof. Hamilton's view.

It is much to be desired that in any future case of a similar nature, a careful histological study of fibres both above and below the thalamus should be made.

It is certainly remarkable that so little degeneration should cross the corpus callosum in cases of even old and extensive destruction of the "motor" cortex; but it is by no means as yet proved that the nutritive centre of its fibres is below. Probably it will be found that the fibres forming this structure are of mixed origin and function.

Postscript.

Since the above paper was written, another case has been reported by Francis and Starr in the American *Journal of the Medical Sciences*, June, 1895.

The patient was a woman aged 45. The duration of the case was fifteen months. During the first nine months the only symptoms were four attacks of general epileptiform convulsions. At the tenth month there was vomiting for a week, and after this progressive mental and physical exhaustion set in. By the twelfth month there was great mental dulness, she was too weak to sit up, was totally blind and had double optic neuritis. The head was persistently deviated to the right, the right pupil was dilated

[1] *Med. Chi. Trans.*, vol. lix.

Name of Observer.	Age and Sex.	Duration of Symptoms.	Tumour Symptoms.			Mental State.	Convulsions.
			Headache.	Vomiting.	Optic Neuritis.		
Bristowe	♂ 46	14 weeks	The first symptoms	Absent	Not stated	Gradual failure, drowsiness. No excitement	Absent
Bristowe	♂ 41	5 weeks	Moderate	Absent	Present	Slow growing stupor	Absent
Bristowe	♂ 51	12 weeks	Absent	Absent	Absent	Increasing drowsiness	Absent
Glaeser	♂ 72	9 weeks	Absent	Absent ?	?	?	Absent
Glaeser	♀ 30	12 weeks	Slight	Absent	?	Some loss of memory	The first symptom repeated, at first general then left-sided, but head turned to right
Glaeser	♂ 60	4 months	Severe at times	Frequent	Present	Irritability, gradual failure, delirious mania	Absent
Berkley	♂ 45	Some years	Absent	Absent	Absent	Insanity, monomania the only symptom	Absent
Oliver	♀ 43	3 years	Absent	Absent	Absent	Visual hallucinations	Absent
Ransom	♀ 24	2 years	Severe at first, afterwards absent	Occasionally after a fit	Present	Excitability, mania. Great improvement for 2 months before death	Early and frequent, mostly left-sided, head and eyes turned to left
Bruns	♂ 77	2 years	Present	Absent	Absent	Progressive dementia with occasional mania	Absent
Bruns	♂ 49	3 months	Absent	Absent	?	Dementia, irritability, symptoms suggesting general paralysis of the insane	Absent

Note.—The fourth case of Dr. Bristowe and the third of Bruns are not included in this list,

Motor Power.	Sensation.	Reflexes.	Cranial Nerves.	Mode of Death.	*Post-mortem* Examination.
Right hemiparesis	Slight failure of vision?	Right knee-jerk increased	Normal	Coma. Fever on day of death	Tumour involving nearly whole of corpus callosum and reaching to left internal capsule
Right hemiplegia, with rigidity, aphasia	Slight right hemianæsthesia	Right knee-jerk increased	Unequal pupils, varying	Coma, with fever for four days	Growth in anterior half of corpus callosum spreading into corresponding part of hemispheres, more into left, internal capsules free
Left hemiparesis, but right limbs also weak	Normal	Normal	Normal	Coma, with slight fever just before death	Sarcoma on anterior $\frac{3}{4}$ of corpus callosum and fornix and invading each centrum ovale above corpus striatum, slightly more extensive on right side
Rigidity and weakness of legs, more of left	Normal	Normal	Normal	Bed-sore and broncho-pneumonia	Tumour = small apple, in genu of corpus callosum invading white substance of hemispheres
Left hemiplegia, but frequent spasms of paralysed limbs	Normal	?	Normal	Very sudden	Large tumour of corpus callosum growing far into right hemisphere
Sudden paresis of right leg, followed by face and arm of same side	?	?	Normal	Coma	Tumour of hinder end of corpus callosum growing very slightly into right and rather more into left hemisphere
Normal	Normal	Normal	Normal	Œdema of lung	Tumour = filbert just behind genu of corpus callosum. Wasting of frontal lobes
Normal	Amblyopia in left eye; colour sense impaired in right, smell lost; taste impaired. Hearing impaired in left ear	Normal except left pupil immobile to light	Normal except for left pupil	Gradual coma	Tumour of genu corpus callosum passing on uncinate gyri, and downwards on optic nerves
Normal	Normal	Normal except after a fit	Normal	Sudden	Tumour = Tangerine orange growing from middle of corpus callosum and pressing on hemispheres slightly
Right hemiparesis with rigidity; face not involved	Partial analgesia on right arm and leg	Normal	Normal	Cerebral haemorrhage	Gliosarcoma, 3 cm. long, just in front of splenium, bulging slightly into right precuneate lobule, and rather more into left central convolutions; gyrus fornicatus free. Left optic thalmus somewhat atrophied. Atheromatous arteries, haemorrhage in lateral ventricles; senile atrophy of cortex
No paralysis or spasm. Speech thick	Absent	Normal	Unequal pupils	Cerebral haemorrhage	Tumour of splenium infiltrating both precuneate lobules and growing into posterior cornua of lateral ventricles. Also growths in right temporal lobe, right Island of Reil, and right optic thalamus. Multiple capillary haemorrhages

as in each the lesion of the corpus callosum was but a part of more extensive disease.

and there was right internal strabismus. There was no other paralysis and no affection of general sensation. The knee-jerks were normal. Headache was throughout slight. Increasing stupor led to death.

At the autopsy was found a large glio-sarcoma rising from the anterior knee of the corpus callosum and infiltrating the greater part of each frontal lobe. It did not appear to compress the optic chiasma.

It will be noted that this case differs from most of the others in the affection of ocular nerves.

Although it apparently grew from the corpus callosum, its subsequent extension makes it more correctly spoken of as a tumour of the frontal lobes.

MULTIPLE TUMOURS OF THE BRAIN: FIBRO-CYSTOMA OF PONS AND CEREBELLUM AND MULTIPLE FIBRO-PSAMMOMATA OF DURA, PIA-ARACHNOID AND CORTEX CEREBRI.

BY F. W. LANGDON, M.D.

Neurologist to the Cincinnati Hospital, and to the Ophthalmic Hospital of Cincinnati.

I saw Miss X., aged 32, brunette, by request of Dr. W. O. Smith on January 15, 1895. Her father and mother were living at advanced age and in good health. Two brothers and two sisters, all of adult age, were living and in excellent health. Two sisters died in infancy; cause unknown. No history of nervous disease or of tumours anywhere in the family.

Personal History.—Patient is one of twins, both girls. She has had measles and perhaps other children's diseases, otherwise enjoying good health until seven years ago, when she experienced a severe attack described as "cholera morbus." Was sick several days at this time and since then has had more or less ill-health, described as "nervous trouble," for which she has consulted various physicians. In November, 1890, she consulted Dr. S. C. Ayres, to whom we are indebted for the following notes: "I first saw Miss ——, in November, 1890. She was deaf in both ears. It seemed to be a case of chronic aural catarrh. There was no obstruction of the Eustachian tubes and but little alteration in the appearance of the drum membrane. Treatment did not relieve her and was not continued long. She had at this time sick headaches. I examined her refraction and found that she had compound hypermetropic astigmatism in both eyes, and gave her correcting lenses; her vision was perfect. On February 27, 1892, I saw her in consultation with Dr. Zenner on account of a marked papillitis of both eyes. Her vision was 0·9, right, and 0·7, left. Two days later I saw her again, and then there was a remarkable and unusual phenomenon in the right eye. In looking at the test card, vision would drop from 0·7 to 0·4 and then in a

moment or two rise to 0·7 again. I watched this change for some time while she remained in my office. No such change was present in the left eye. A perimetric examination showed that the upper part of the field of vision was contracted and also the field for red. I did not see her again until January 28, 1895; she was then suffering so much pain that I could not test her accurately, but I found that her central vision was normal as she could read fine print with either eye. I found, however, that there was paresis of the right abducens, with diplopia on the right side."

Patient states (January 15, 1895) that she had within a few months past, several attacks of vertigo and vomiting, apparently brought on by sudden movements, as turning in bed, &c. Says she *feels nausea before vomiting*. Also complains of numbness of left extremities and left side of body.

Clinical History.—Patient in bed, but has been up and about room more or less daily. Decubitus dorsal. Temperature under tongue 99·4 F.

Nervous System.—Mental state normal; memory excellent; patient converses and writes with intelligence and manifests much interest in her own case. Speech somewhat slow but unhesitating and correct. Handwriting steady, uniform and legible; letters well formed, words correctly spelled and sentences properly constructed.

Sensory Symptoms.—She complains of a general sense of fatigue and excruciating paroxysmal pains in the head, chiefly in right parietal and occipital regions, from which she derives relief by assuming an opisthotonic posture (resting head on vertex). Examination also reveals hyperæsthesia of the scalp over the right frontal, parietal and occipital regions. Tactile paræsthesia of slight degree over left face, body and extremities.

Vision: Pupils moderately dilated, responsive to light. Patient complains of double vision at times, but reads and writes without apparent difficulty. (See previous report.)

Hearing markedly impaired on both sides, practically absent on left. (See also previous report.)

Taste and smell not tested, but no deficiency or perversion noted by patient.

Motor Symptoms.—Left hemiparesis of face and limbs present, and patient says she tends to turn toward left side in walking. Tongue protruded in median line. Later observations (within a month) by Drs. P. S. Conner and W. O. Smith, record a marked deviation to left, which disappeared within a few days to return

later. During the last few days of life, marked symptoms of bulbar paralysis developed—as evidenced by great difficulty in swallowing and articulation.

Patellar and olecranon reflexes not notably altered.

Heart sounds weakened, no bruit detected. Pulse 84 to 90, soft, quick stroke.

The other systems were normal, and urinary analysis showed no abnormal constituent.

The salient features of the case at the time of my first and only visit (thirty-nine days before death) were:—Violent paroxysmal pains in head, hyperæsthesia over right side of scalp, diplopia, bi-lateral deafness, left hemiparesis and vomiting *with nausea*, added to which were the optic neuritis and abducens paralysis, noted by Drs. Zenner and Ayres. Upon these I based my opinion of a tumour at the base of the brain, probably pressing on the pons varolii and involving the fourth, fifth, seventh and eighth nerves or their nuclei. From these symptoms, especially the left hemiparesis, I supposed that the growth was in the upper half of the pons on the *right* side, whereas its actual situation discovered after death was over the lower half and *left* side.

It was plain that no one cortical lesion would account for all the symptoms presented; and the absence of convulsions, monoplegia or mental impairment excluded, to my view, an extensive or multiple cortical involvement. Further, the evidence pointed to disturbance of the right fourth and sixth nerves (or their inter-communications) to the right fifth, left seventh and both eighth nerves also; and only in the region of the pons was this possible to occur from a single lesion, without loss of consciousness.

The vomiting and rotatory gait would naturally point to the cerebellum, but might easily be accounted for by irritation of its peduncles by a growth in the pons.

Report of Autopsy, February 28, 1895, at four o'clock, p.m., five days after death.

Body well preserved; no embalming fluid or other preservation used. Rigor mortis present to a slight degree. The head only was examined. Calvarium removed in the usual manner. Dural outlines and general consistence of

brain normal. Dural vessels markedly hyperæmic, the larger meningeal trunks standing out as prominent dark blue cords. Cortical veins appear through dura fuller and more distinct than normal.

The lower border of falx cerebri at a point 6 cm. posterior to the crista galli contained an ovoid neoplasm, the size of a kidney bean, enclosed within the layers of the falx; the left layer, however, had undergone absorption, so that the fibrous capsule was deficient over the central two-thirds of the growth on the left side. Immediately posterior to this growth the falx in the region of the inferior longitudinal sinus, for the space of 25 mm. longitudinally by 5 mm. vertically, presented a recticular or cribiform appearance apparently the result of imperfect development or absorption; but this bore no direct relation to the growth mentioned, nor to any other.

A second neoplasm, half the size of the first, of similar appearance but more flattened, was situated at the superior margin of the falx, slightly posterior to the first mentioned growth, with which it was connected by a narrow isthmus.

Three similar growths, also more flattened than the first projected from the inner surface of the dura of the convexity over the posterior extremities of the first and second frontal convolutions. These measured respectively 3 mm., 6 mm., and 20 mm., in greatest extent and from 2 to 3 mm. in thickness. A sixth neoplasm, ovoid in form and the size of a large kidney bean, projected from the under surface of the dura—but still enclosed between its layers—immediately to the right of the great longitudinal sinus, which it did not invade. This growth occupied a position nearly corresponding with the upper extremity of the Rolandic fissure.

A seventh growth, also the size of a kidney bean, was enclosed in the meshes of the pia, just anterior to the *lamina cinerea*, where it bridged the great longitudinal fissure and penetrated slightly the cortex of both frontal lobes (*gyri recti*).

The eighth and principal tumour (fig. 1) came into view on dividing the *tentorial* attachment to the petrous margin. Its anterior extremity, the size of the end of the little finger,

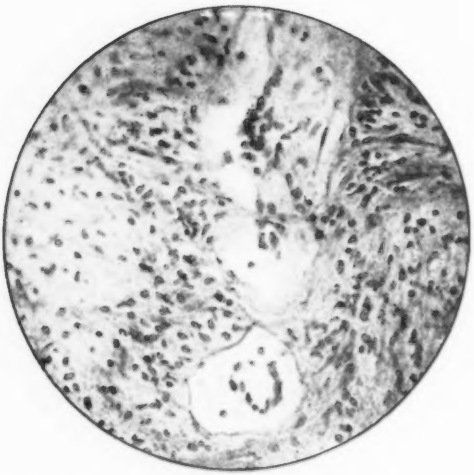


FIG. 2.

A section of solid portion of main tumour (No. 8 in report). x 320. Photo-micrograph by Dr. M. H. Fletcher.

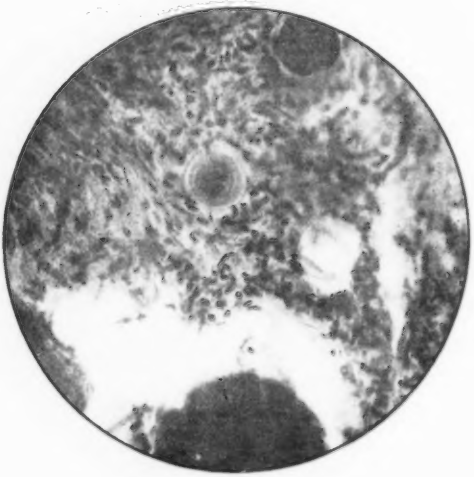


FIG. 3.

Section of tumour from falx cerebri (No. 1 in report) showing concentric laminated nodule near centre of field. x 320. Photo-micrograph by Dr. M. H. Fletcher.

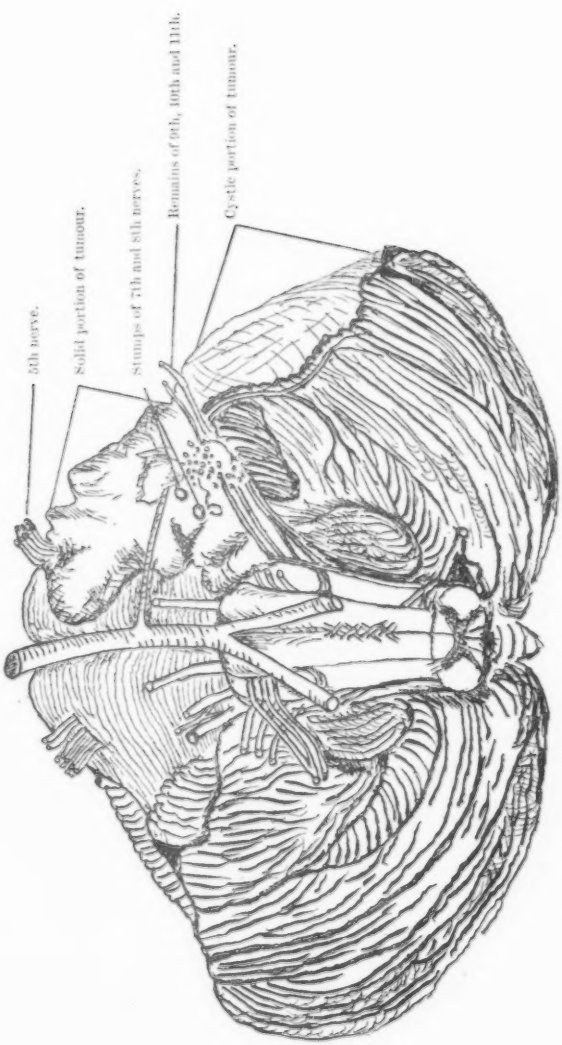


FIG. 1.

Sketch of principal tumour and surroundings—natural size, made immediately after removal.

projected from the under surface of the pons on the *left side.*

As the entire tumour came into view, it was seen to be a firm, irregularly nodular, yellowish, encapsulated mass, in bulk about equal to an English walnut, but more flattened and irregular in form.

This growth involved the left lateral half of the pontine surface—for its posterior two-thirds—and also extended outward and posteriorly, invading the antero-lateral margin of the left cerebellar hemisphere. Extending still further in the same direction, this solid portion of the growth was continuous with a cystic portion, thin-walled, evidently composed of pia and arachnoid, and containing about half an ounce of clear, amber-coloured, somewhat viscid fluid; a small portion of this fluid was decidedly gelatinous in consistency. The left cerebellar hemisphere, where compressed between the growth and the tentorium was softened and semi-diffluent.

The left half of the pons (its posterior two-thirds) was compressed and atrophied by the growth to about one-half its normal lateral extent (on its inferior surface) and *the pons as a whole was displaced to the right.*

The left flocculus was distorted, flattened and continuous with the under surface of the growth.

The considerable segment of the left cerebellar hemisphere that was atrophied and absorbed or softened by the pressure, amounted perhaps to one-fourth of the total bulk of this hemisphere.

Except as described the external appearance of the cerebellum was normal.

The cranial nerves in *apparent* relation with the growth, were the left fifth which deeply grooved its anterior (superior) surface for a distance of 11 mm., and apparently terminated in the tumour substance as a yellowish, moderately firm, mixed mass of gelatinous substance and nerve fibres. Tracing the nerve anteriorly from the growth it passed as a similar bundle of mixed gelatinous and normal nerve fibres, to the Gasserian ganglion, beyond which the fibres and divisions appeared normal. The above described conditions involved both roots of the left fifth nerve.

The left sixth nerve, somewhat displaced mesially at its origin by lateral pressure on the pons above referred to, passed forward in the groove formed at the junction of the pons and the neoplasm. It was normal in appearance and not invaded by the growth.

The left seventh and eighth nerves were not visible at their usual superficial origin since this was occupied by the growth, which is here apparently continuous with the pons and oblongata substance.

Two rounded stumps are apparent on the surface of growth corresponding to their usual course, from which the nerves have probably been torn in removing the specimen. (See fig. 1.)

The ninth, tenth and eleventh nerves emerged normally, traversed the under surface of the growth for 20 mm. and partly entered its substance, a few fibres, mainly of the tenth nerve traversing the growth, emerging laterally and continuing their course towards the jugular foramen.

The remaining fibres, by far the larger bulk of these nerves, had softened and disappeared upon reaching the growth; and in this immediate region of disappearance of nerve fibres the surface of the growth is broken into miliary nodules, perhaps a hundred in number.

The left twelfth nerve emerged normally and was not directly compromised by the growth, but was probably subject to mechanical pressure at times during life.

The left anterior inferior cerebellar artery, as shown in fig. 1, traversed the lower surface of the growth for 19 mm., penetrated its substance for 13 mm., and emerged to resume its normal distribution, apparently not infiltrated or compromised in any manner by its intimate connection with the tumour. On section the cerebral cortex and alba were of normal appearance and consistence. The lateral ventricles were markedly dilated, their anterior and posterior cornua readily admitting three fingers; choroid plexuses fringed with numerous clear round cystic dilatations, miliary in size, containing a pellucid fluid.

A brief analysis of the symptoms in the light afforded by the autopsy, may be of interest from the standpoint of

localisation of function. At the outset, our attention is naturally directed to the relationship, if any, between the supposed attack of "cholera morbus" seven years previous to death and the pathological process in the brain. Was the attack of vomiting and diarrhœa to be looked upon as being in any sense an ætiological factor in the disease? Or was it symptomatic of an early stage of the growth in the cerebellar peduncles or the vagus nuclei and trunk? The latter would seem the more probable explanation to my mind.

As regards the area of nervous substance involved, the symptoms presented were less marked up to the last few days of life than might have been expected, having in view only the ordinary facts of localisation. These comparatively slight effects of so serious a lesion are to be accounted for probably by the extremely gradual progress of the growth, allowing time for a considerable degree of accommodative adjustment on the part of the tissues involved. This, however, eventually reached its limit, and hence the sudden development of marked bulbar symptoms toward the final end.

The cranial nerves and nuclei involved symptomatically correspond with the anatomical situation of the growth, as may be seen by our diagram (fig. 1), with the exception of the *fourth* and *fifth* nerves. While ocular examination by Dr. Ayres revealed an apparent paralysis of the fourth, its trunk was not directly involved in any part of its course. Hence the diplopia, which was the apparent result of its impairment, must probably be attributed to the involvement of the co-ordinating fibres (*posterior longitudinal bundle*) by which harmony of action is brought about between the nuclei of the third, fourth and sixth.

It will be noted that, though the fifth nerve was seriously compromised in structure, some symptoms that might have been expected, viz., hyperæsthesia, anæsthesia, and trophoneuroses of *left face* and its organs, with spasm, paresis or paralysis of muscles of mastication, did not occur, as sufficient fibres to carry on the function evidently escaped degeneration.

We are also confronted by the clinical symptom, in this

case, of *right-sided hyperæsthesia*, over the forehead and scalp. Since the main trunk of the *left* fifth was the one apparently irritated by the growth, the hyperæsthesia would naturally be expected to be referred to its terminal distribution. The explanation of this apparent discrepancy in localisation may be due to the fact that the tumour by its pressure exerted from the left side toward the right may have pressed the right fifth nerve against the acute margin of the tentorial attachment to the petrous bone, and so given rise to the symptoms mentioned.

The bi-lateral deafness, most marked on the left, was evidently due to two factors: (1) The direct involvement of the left acoustic nerve by the growth; and (2) Pressure on the pons affecting the fibres from the right nuclei in their course toward the cortex, through the pons, crus and internal capsule.

The absence of complete left facial paralysis (there being only paresis present) is explicable by the fact that some fibres of the *portio dura*, though surrounded by the growth, maintained their power of conduction, as was also the case with the greater portion of the glosso-pharyngeal, vagus and accessorius up to the last few days of life.

The left motor paresis was apparently due to pressure on pyramidal fibres *after* their crossing.[1]

It is evident, moreover, that a very limited lesion of the *right upper* half of pons (above the facial decussation) would produce practically the same motor symptoms as here presented by our tumour in its *lower left* segment.

[[1] In the discussion which followed the reading of this paper before the American Medical Association at Baltimore, May, 1895, Dr. Charles K. Mills and Dr. H. T. Patrick very pertinently directed attention to the fact that it was difficult, even with the specimen before us, to account for the motor and sensory paresis existing on the same side as the tumour, since the latter was above both the motor and sensory decussations. It was this circumstance also which led me in the first instance (*ante-mortem*) to locate the lesion on the right side of pons. After a careful weighing of the matter in all its aspects, I have concluded that the only rational explanation, in the light of our present knowledge of the subject, is that the motor paresis and sensory paræsthesia were due to a "*contre coup*" effect, *i.e.*, of pressure by the tumour of the right periphery of the medulla against the comparatively acute margin of the foramen magnum at a point above (before) the motor decussation and also above (after) the sensory (fillet) decussations. The left *facial* (motor and sensory) symptoms, on the other hand, are sufficiently accounted for by the direct involvement of the tri-facial and the portio dura by the tumour.—(L.)]

It is worthy of note that the vomiting, which occurred chiefly or entirely upon sudden movement, was *preceded by nausea*, contrary to the rule in cerebellar vomiting. This would perhaps indicate that the *involvement* of the vagus trunk or its nuclei was the cause of the nausea and vomiting.

We may also note the protrusion of the tongue to left side (left paralysis) and its apparent recovery within a few days. If due only to direct pressure of the solid growth a right-sided tumour pressing on *upper half* of pons would also account for this symptom, by involvement of cortical tract to medulla. In this case, however, the symptom would have been permanent, whereas its sudden disappearance is accounted for by fluid pressure on the hypoglossal trunk (shifting of fluid in cystic portion of growth).

One more symptom deserves mention. The patient derived great relief from the severe intra-cranial pain by assuming a semi-opisthotonic position, resting head on vertex. It is evident that by this position the weight of superimposed brain mass was removed in a measure from the growth, and especially from the left fifth nerve, which in the ordinary position of head was compressed between the tumour and its solid backing of dura and bone below, and the brain above.

Therapeutically, Dr. Smith informs me, the greatest relief to pain was derived from *antipyrine*, its effects being superior even to morphine in this case, and without disagreeable accompaniments.

Pathological Report on the Tumours.

BY ARCH. I. CARSON, M.D.
Curator to the Cincinnati Hospital.

Microscopical Examination.

Two small pieces from different peripheral portions of the main tumour (No. 8 in report) were examined. Both specimens show practically the same conditions; that is, fibroid material arranged irregularly in intercrossing fasciculi

and in some areas in whorls. One specimen was of quite close texture and not very vascular. There were present numerous small bodies which stained most deeply, round, oblong, and spindle-shaped; evidently nuclei of connective tissue cells and embryonal cells.

The second specimen was of a much looser texture and more vascular. It showed similar nuclei, but very much fewer spindle-shaped ones. The fibres were not so frequently arranged in bundles, but were interwoven in all directions. The vessels, around which the fibres were arranged more or less concentrically, were filled with red blood cells. (Fig. 2.)

From the examination of these specimens, from two different parts of the tumour, it is concluded that the growth is a fibroma.

There was also examined a small mass (tumour number one in report) ovoid in shape, measuring 10 mm. in length, 7 mm. in width, and 4 mm. in thickness, which was removed from about the centre of the falx cerebri. This mass under the microscope showed a highly cellular fibrous tissue, containing numerous concentrically arranged chalky concretions, the latter characteristic being evidenced in the cutting of the sections. It is concluded that this mass is a psammoma. (Fig. 3.)

A CRITICAL STUDY OF A FEW OF THE CHANGES FOUND IN THE FIELDS OF VISION, TAKEN WHILST THE EYES ARE PLACED AT RIGHT ANGLES TO THEIR ORDINARY POSITION.

BY CHAS. A. OLIVER, A.M., M.D.

Attending Surgeon to the Wills Eye Hospital; Fellow of the College of Physicians of Philadelphia; Member of the American and French Ophthalmological Societies.

THE original suggestion as to this character of work was kindly given to the writer by Dr. S. Weir Mitchell, of this city, in the fall of 1893, during a personal conversation upon various matters pertaining to physiological optics, colour-vision, &c. Dr. Mitchell desired to know why it was that artists could obtain more vivid impressions of the colours of a distant landscape whilst the head was lowered into a horizontal position. After hazarding, as a part explanation of the reason, that a greater blood supply was thus offered to the organ when the head was held in this way, and that there might be some difference in the combined colour values when refracted through another series of meridians of the dioptric apparatus, the writer concluded that a most interesting, and perhaps valuable, series of experiments upon the subject might be established amongst the abundant clinical material at hand, in one or more of his public services, and amongst his more intelligent private patients and his friends.

Limiting the present communication to those cases that gave full acuity of vision, had normal fields of vision, and showed healthy eye-grounds, a great number of observations were made by both the writer and several of his most com-

petent friends and assistants.[1] In every instance, as far as practicable, the two series of experiments were performed under the same conditions. After the fields for white, yellow, blue,[2] red, and green were gotten in the ordinary manner, the patient or subject was placed upon a table or a sofa, so that the temporal or outer side of his eye was situated either superiorly or inferiorly. When in one of these positions, either before the perimeter or the gauged blackboard, and by both natural and artificial light, the same order of field-taking was instituted and consummated in various ways.

With the greatest precaution as to time of day, similar character of illumination for related experiments, and proper rest of the organ, the following observations were rendered sufficiently certain for publication.

(1) Equal areas of the same colour held at equi-distant points around the fixation object, when the fields of vision are taken in their ordinary positions, are more deeply saturated and more intense in vividity when they are situated at the temporal side of the point of fixation;[3] the relative positions of the order of brightness being temporally, inferiorly, nasally, and superiorly.

(2) Unequal areas of the same colour held at equi-distant points around the fixation object show, when the fields are obtained in the ordinary manner, that the smallest colour areas are more distinctly visible and more deeply saturated to the temporal side of the point of fixation; the relative position of the necessary increase in colour area, to give the same vividity as that seen to the temporal side, being inferiorly, nasally, and superiorly.

(3) Equal areas of the same colour, studied during the method of ordinary field taking, are sooner perceived and

[1] The writer here acknowledges his indebtedness for valuable help in this work to Drs. William Zentmayer, William Campbell-Posey, Frederick Krauss, Barton F. Potts, and Frank Savary Pearce.

[2] In many cases yellow and blue were omitted. In a large number of private subjects, however, yellow and blue were tried sufficiently often to admit of their entrance into the series.

[3] In some cases, the best point of excentric colour vision seemed slightly down and out from the point of macular fixation—*i.e.*, just beneath and between the fixation point and the physiological blind spot.

are brighter in tint when they are approached from the temporal periphery to the fixation object, than they are when they are brought in from any other peripheral border of the colour field; the order of greatest distance from the point of fixation in the different parts of the fields before the colours are recognised and properly named as such, being first, to the temporal side; secondly, to the inferior side; thirdly, to the nasal side; and fourthly, to the superior side.[1]

(4) Unequal areas of the same colour approached to the fixation point from the various peripheral borders of the fields of vision taken during the ordinary method of field-taking, show that the smallest colour areas are sooner perceived and become more quickly saturated, when they are brought in from the temporal side; the inferior and nasal sides being the next respectively and the superior side the last.

(5) In every instance where the fields of vision were obtained at right angles to the ordinary position, by placing the temporal field either superiorly or inferiorly, both equal and unequal areas of the same colour approached from the various peripheral borders of the fields of vision to the fixation point, show the same relative order of betterment of comparative sizes, positions, and saturations of tint, in that portion of the field which has been previously the temporal one.[2]

Conclusion.

As a logical deduction from these observations, and as a partial answer, at least, for the reason of the empirical method of dipping the head at right angles to its ordinary position, so frequently made use of by artists in order to

[1] When care was taken to see that equivalent amounts of light were obtained in all meridians, the relative shapes of the fields of vision were not changed whilst the fields were tried at various angles.

[2] In fact, numerous experiments made since, which have no direct bearing upon the exact title of this paper, and which, with other important and interesting observations, will be reserved for a more extended communication, distinctly show that this is true, no matter how the temporal field may be placed.

obtain a better colour effect whilst observing a distant view, it seems most probable that the cause is, that by this plan, as it were, a stronger and a more highly evolutionised series of sensory elements are made to receive natural impressions which, under ordinary circumstances, are received and recorded less vividly by a series of lower grade and less powerful structures.

A CASE OF SYMPATHETIC PAIN: PAIN IN FRONT OF THE CHEST INDUCED BY FRICTION OF THE FOREARM.

BY T. K. MONRO, M.B., M.S., GLASGOW.

THE patient, who was a fireman, aged 44, consulted me in the Spring of 1894, at the dispensary of the Royal Infirmary. On account of the curious nature of the case, I afterwards called at his house, traced out the important areas on the surface of the body with pen and ink, and then photographed him. The accompanying illustration is taken from the photograph.

The following are notes of the case (April, 1894):—Patient complains of pains in his chest and right arm. He is disposed to attribute them to a "cold" which he got three and a half months ago, while sweating profusely at his work. (He had no cold in the head, however; and, indeed, apart from pain in the chest, no symptoms of cold at all, so that his explanation is unsatisfactory, and it will be seen hereafter that injury was a more probable cause.) Excepting an attack of fever last summer, he never had a day's sickness till his present illness began. The pain came first into his right "shoulder," at the back. It began suddenly, whilst he was firing in the stokehole, and compelled him to stop work immediately. The ship's surgeon told him to lie on his back without turning. That pain has never left him since, though it varies in severity. It does not awake him at night, but it often prevents him from falling asleep. It is seated in an area (B—not seen in the figure) on the right side of the back of the chest, at about

the level of the seventh dorsal spine. It is rectangular in shape, and nearly square, measuring an inch or two each way. The most painful spot is at the centre, where there can be felt what seems to be a localised thickening of the rib, as if there had been a fracture or other local injury. The continuous pain in this area B is variously described as a "dead burning heat," "like red-hot sand or iron passing through the flesh," or as if a knife were driving straight in. At first it extended up to the back of the head, but it does not do so now. It sometimes passed down to the small of the back, and then up again.

On the right side of the chest, in front, there is an area (F—marked out in the figure), bounded laterally by a vertical line extending from the axilla down to the sixth rib or thereabouts. The mesial boundary of this area is about a couple of inches nearer the middle line, and just external to the right nipple. Below the right nipple there is a spot which is sometimes painful (indicated in the figure by a cross). In the area F, "tearing pain" is felt. Occasionally, but only in consequence of exertion, *e.g.*, quick walking, or turning suddenly in bed, a momentary pain shoots across from this area towards the left side, about as far as the vertical line of the left nipple. (In the figure, the vertical line on the left front indicates the limit towards the left beyond which this shooting pain does not pass; the horizontal line shows its usual level and direction.) The momentary shooting pain is generally followed by a feeling of "pins and needles," lasting for five or ten minutes, at a point just below the right nipple.

A third painful area to be described (A) is on the posterior aspect of the right forearm (shown in the figure). Pain here is specially apt to be induced by pressure over the radio-humeral articulation. The area of pain extends slightly higher than this, and includes the middle two (longitudinal) fourths of the dorsal aspect of the forearm; it reaches as far as the four inner metacarpo-phalangeal articulations. The five digits, however, are free from pain. The surface A is about an inch broad at the middle of the forearm, which is itself two and a half inches in breadth at

this part. A narrows at the wrist and widens again towards the bases of the fingers. When pain is induced at the elbow-joint, it is always severe; it may be associated with slighter, but occasionally with quite as severe pain further down the limb. About a month ago, the pain in the right elbow was so severe that the limb was practically useless. Bending the elbow causes severe "cutting" pain just above the external condyle. Flexing the wrist also causes a slight, but not nearly so great pain at the elbow. The joints are never swollen.

One of the most remarkable features of the case comes now. *A touch on any part of A* (but not on any other part of the forearm) *is felt in F* (chiefly at its middle part), *in addition to being felt in A*. The converse is not the case. *Firm pressure on A* (if the region of the elbow be avoided) *causes no pain in A, but causes severe "tearing" pain in F* (chiefly at its middle portion).

F is the only part of the trunk in which pain is induced by handling the arm. Rubbing the dorsum of the forearm induces the pain in the chest; indeed, the patient seems to have first discovered this sympathetic pain on one occasion when he was washing his arms, about a month after the accident to be presently described. He says it is much worse when he uses cold than when he uses warm water for washing.

Extension of the right wrist causes no pain at the elbow, but flexion of the fingers causes so much pain as to prevent him from gripping objects with his right hand. There appears to be no tenderness of the nerve-trunks in the forearm.

There are pains occasionally, but only for a second at a time, in the knees and ankles; these pains very seldom occur in the left knee, and are not so severe there as in the right knee. Apart from this, the left leg is perfectly well, and the left arm and left side of the trunk are healthy. There is no weakness of the legs; the ground feels naturally to the feet; the knee-jerks and the pupils are normal. There is no nystagmus, ataxy, headache, or vomiting. The bowels are regular. There is no *tache cérébrale*. The heart and lungs seem healthy, and there is no evidence of

DESCRIPTION OF FIGURE.

The area A is seen marked out by ink on the back of the right forearm and hand. A tattooed figure is seen on the upper arm. The area F is marked out on the right side of the chest, and a cross below the right nipple indicates one seat of pain. Friction or simple pressure over A causes pain in F.

aneurism. The pectoral muscle-fibres are more irritable when struck on the right than on the left side. There is no obvious atrophy of the muscles in the right hand. Patient says he has never had rheumatism. He has had no cough or spit in the course of his illness, but he breathes cautiously, because a deep breath is apt to catch him in the right side.

His chest was extensively blistered by a doctor about four months ago. The blistered surface healed within a few weeks. One doctor told him he had "pleurisy," another said "dry pleurisy," and a third said he had "pleurodynia."

Careful inquiry reveals facts that assist greatly in explaining these curious symptoms. About eight days before patient became ill, he stepped upon an insecure plate in the engine room, and lost his footing. The result was that he struck his elbow (? A) against something which caused his elbow or fist to strike against his chest in front (? F); after which his body was twisted round so that his back (? B) knocked against a corner. He subsequently felt some soreness about B, but he did not think—at the time, at least—that anything was broken.

Patient states that for years, *i.e.*, since long before his illness began, he has sweated greatly, and particularly in bed, with his right side only. This side, as he has noticed hundreds of times, will be soaking when the left is dry. He may find his right ear filled with perspiration. The right side perspires in this way all over, but he says that both sides sweat alike when he is at work.

When a patient complains that rubbing his arm causes pain in his side, it is perhaps natural that the idea of allochiria, as described by Professor Obersteiner, or of allachæsthesia, as described by Sir Grainger Stewart, should occur to the mind. This case, however, does not conform strictly to either of these types as originally portrayed. At the same time, it must be remembered, different writers on allochiria have shown that one particular form of sensation (touch, pain, temperature, tickling, sight, hearing) may be affected by itself. If the present case is

allied to allachæsthesia (rather than to allochiria, where the sensation is referred to the opposite side of the body), pain is the form of sensation involved; for pressure or friction on the forearm, which is not such as to cause pain locally, causes it to be felt in front of the chest.

On the more convenient hypothesis that the case is an example of sympathetic neuralgia, the nerve-connexions between the two parts become interesting and important. In normal individuals, the area A in the forearm is supplied by the lower external cutaneous branch of the musculo-spiral nerve and by the posterior branch of the musculo-cutaneous. As Ross has pointed out, the cutaneous nerve-supply of this region is homologous with that furnished in the trunk by the dorsal portion of each anterior primary division of a segmental spinal nerve, including, therefore, the lateral cutaneous nerves of the thorax. The figure shows that the area F on the surface of the chest must be almost entirely supplied by these lateral cutaneous branches. As the forearm, however, is innervated from the cervical cord, and the particular part of the chest-wall in question from the dorsal cord, at a considerably lower level, the special path of connexion between the chest and the forearm in this case is not very easy to comprehend.

Reviews and Abstracts.

Entwurf zu einer physiologischen Erklärung der psychischen Erscheinungen. Von Dr. Sigmund Exner, o.ö. Professor der Physiologie und wirkl. Mitglied der kais. Akademie der Wissenschaften zu Wien. I. Theil. Mit 63 Abbildungen. Leipzig und Wien. F. Deuticke. 1894. Pp. viii., 380.

The "explanation" of a scientific phenomenon consists in the statement of the conditions under which it occurs. The psychologist explains a given mental process by analysing it into its simplest conscious constituents, and by referring these to their physiological substrate. An enumeration of the physiological conditions of states of mind is held to be essential by modern psychology: Wundt's conception of "physiological psychology" is practically the same with Fechner's idea of "internal psychophysics."

It is in this, the physiological sense, that the word "explanation" is employed by Professor Exner. "By an explanation of psychical phenomena I mean their reference to physiological processes in the central nervous system already familiar to us in other connections." "The problem which I have set myself is that of the reference of the most important psychical phenomena to differences of degree in the excitatory condition of nerves and nerve-centres, *i.e.*, the reduction of all the [qualitative] variety of consciousness to terms of quantity, and to the diversity in the central connections of nerves and centres which in all other respects are practically undifferentiated." If the work arouses distrust at first sight by the number and prominence of "unproven, hypothetical propositions," we must remember that principle, rather than detail, is at stake. It is enough to have shown that mental processes are capable of scientific treatment; particular hypotheses may be looked upon with comparative

indifference. At the same time, the author realises the necessity of basing hypothesis, wherever possible, upon a foundation of ascertained fact, and brings to his task of explanation the interest and experience of a quarter of a century.

The book is not written for the general reader, but for those who have mastered the elements of physiology or psychology. To students of the latter science it makes a very special appeal. They are accustomed always to regard the psychical as primary, and the physiological as secondary: their analysis begins with the mental. Professor Exner reverses this order, and passes from the nerve process to the correlated conscious process. Psychology, in his series of chapters, takes on a new appearance: he begins with voluntary movement, and goes on to deal successively with attention, sensation, perception, idea, and the phenomena of intelligence. A second Part is to discuss more special topics—the relations of the separate divisions of the central nervous system to the various mental functions. It is not to be expected for some years to come.

Chapter I. is a preliminary exposition of the anatomy of the central nervous system. It is intended to assist psychological readers in their understanding of the book, and is well adapted to its purpose. Though very greatly condensed, it is clear and simple in statement, and covers the most important facts in an adequate way.

Chapter II. gives the physiological data and inferences (*die physiologischen Grundphänomene*) upon which subsequent theories are based. The author says modestly that it "contains nothing new." But it has the great value—apart from its function as the foundation of the whole work—of being the first attempt at a general physiology of the central nervous system. It is undoubtedly coloured to some extent by the thought of the purpose to which it is to be put; but departures from established fact are frankly termed hypothetical, and nowhere involve any contradiction of physiological laws. The first of the five sections deals with the conduction of excitation within the nerve. The second discusses the central "exchange" (*Umsatz*), "the transference of a stimulus in the grey matter . . . from one known path to another path, of which the existence at least is known," under the headings of reflex movement (reflex time, summation of stimuli, the "explosive" process in the reflex, theory of conduction in the grey matter, the grey nuclei) and concomitant sensations, cases of the "transference of an excitation from one

centripetal path to another." We may notice here that the ganglion-cell is spoken of as "charged" and "discharged"; that fibre masses or ganglion-cells are "related" according to the closeness of their interconnection, anatomical or physiological; and that the cerebral cortex receives the name "organ of consciousness." The third treats of central inhibition (of motor and sensory impulses) and facilitation (*Bahnung*). The latter concept seems destined to play a large part in physiology and psychophysics: it is interesting to compare Professor Exner's illustrations with von Kries' paper upon predisposition (*Einstellung*) in the *Zeitschr. f. Psychol.* (vol. viii., pp. 1 ff.). The two last are entitled "Interaction of Excitation in the Grey Matter" (tetanic reflex action, the physiological identity of the mechanism for tetanic reflex action and for leaping, combination of successive movements) and "Sensomobility," the "capacity of man or animal for movement, influenced, controlled, or conditioned by centripetal nervous excitations" (the true reflex, characterised by subcortical regulation; instinctive movement, regulated by intention—*Intentionsregulirung*—in which the attention effects the temporary installation of a subcortical reflex apparatus, subserving the intended purpose; and conscious movement, with cortical regulation). The processes involved are exemplified by elaborate anatomical-physiological diagrams. The rubrics of the five sections will suffice to give the reader some idea of the terms in which the author has couched his psychophysical explanations. The hundred pages of the chapter hardly admit of any other kind of summary, occupied as they are either with progressive argumentation or with detailed evidence upon particular points.

The transition from sensomobility to voluntary movement (Chapter III.) is an easy one, just because the writer is concerned with the physiological side of the process, and can neglect its psychological *differentia*. "The fact which stamps these actions as voluntary, the fact, *i.e.*, that they are the result of impulses of the will, and as such are performed with consciousness," need not be considered for the time being. Beginning with combinations of voluntary movements, Professor Exner disclaims the view that voluntary motor impulses are directed upon the innervation of a particular muscle group. It is the object, the result of the movement, which is in consciousness. The result is given in terms of sensation; and if sense is impaired, the movement becomes difficult or impossible. Voluntary movements are of two kinds, mixed and pure. The act of

swallowing is taken as an illustration of the former, speech as an instance of the latter. Most of the "voluntary" acts of animals are mixed: cortically initiated, but thenceforward regulated by subcortical centres. In man, subcortical regulation has not disappeared, although it is largely subordinate to cortical. Indeed, the cortex is probably operative in many cases of "unconscious" action, as, *e.g.*, in the innervation of trunk and legs which accompanies the conscious movement of the arm when an axe is lifted to strike,—witness the effect of practice and attention. On the other hand, subcortical influences are to be traced in pure voluntary movements (innervation of unnecessary muscles, symmetrical concomitant movements).[1] The difficulty of executing a voluntary movement for the first time—*e.g.*, when a child is learning to talk—consists "in discovering and hitting the right cortical paths, where there are plainly so many which are closely related to one another." Innervation and inhibition probably co-operate to produce the intended result. In the illustration chosen, the intended result is the production of a simple sound. The mechanism of this sensory regulation, or regulation by idea, differs only in the range of its possible effects from that of the regulation of the instinctive movement "by intention." The child, in the course of performance of numberless voluntary movements, comes by chance or by imitation of mouth-movement to say the particular word. An association is set up in the cortex between the sound and the voluntary impulses leading to it. Repetition strengthens the association, and finally brings knowledge of the muscular innervation involved in the production of the sound. But the cortical association means a relation between the corresponding subcortical centres: repetition of the sound, *i.e.*, simultaneous excitation of the paths for hearing and speaking, sets up an "intercellular tetanus" in these centres. Now, therefore, the idea of the sound, excitation from the sensory end, begins to be important for correct innervation; for "attention to the sense impression" or the "idea" of it acts by way of facilitation upon subcortical centres. The theory is supported by the empirical observation that no movement whose sensory effect cannot be ideated is under the domination of the will.

The following section, on the temporal course of voluntary

[1] The subcortical centres for instinctive movement (regulation by intention) are probably the thalami and corpora quadrigemina; those for voluntary movement, the caudate and lenticular nuclei (Meynert, Nothnagel, Bechterew).

movements, analyses the physiological process in the reaction experiment, simple and compound. The phenomena of facilitation, and the influence of idea on voluntary movement, are visible in the introspective and objective results. The paragraphs are timely, as a contribution to the theory of the reaction, apart from their special bearing upon the matter in hand.

The author passes in Chapter IV. to the consideration of the attention, which has been mentioned before in connection with instinctive movement, and examined under certain aspects in the discussion of the reaction experiment. "We have seen that the act of will produces changes in the nervous system, the effect of which is, that an excitation now follows a path A, and now, when a different change has been voluntarily set up in place of the former one, takes the path B. Changes thus set up have the character of states (*Zustände*). I regard this interaction of different parts of the central nervous system as the essential thing in what we call attention (*Wesen der Aufmerksamkeit*)." The attention, however, is not simply concerned with the interaction of sensory excitation and motor direction; it may be exclusively sensory, though, it is true, there always remains a tendency to movement. In any case, "whether it is turned to sense-impression or movement or memorial image, the attention has a two-fold function: it makes certain paths within the nervous system especially practicable, and holds them open for some time, more or less; and it reduces the conductive capacity of a large number of other paths." This state of the system is termed *Attention* (not *Aufmerksamkeit*). It plainly consists in an "attentional facilitation" and an "attentional inhibition." The latter implies "an increased tonus of the cells, in spite of which their discharge is rendered difficult." The former might be thought to imply an enhancement of the sensation attended to; but Professor Exner shows, by one of his characteristic schemata, that the conscious result of stimulus-increase is different from that of attention-increase. Sense-impressions have been "worked over" at least once (in the ganglia of the caudex cerebri and sensory nuclei) before their impulses are affected by attentional facilitation and inhibition. There are certain centres to which the attention has no access (those for cardiac movement, for the pupillar reflex, &c.). It can influence the results of all movements and inhibitions, the execution of which may be accompanied by consciousness.

This account of the physiological equivalent of attention evidently demands a mixed, positive and negative, psychological theory of the state. Neither a purely positive explanation, like

G. E. Müller's assumption of a "centrosensory reinforcement" of peripheral excitations, nor a purely negative one, like Külpe's "inhibition" hypothesis, is adequate to the facts of nervous function.

The remaining four chapters of the work take up the examination of the contents of consciousness, in the order from simple to complex. Chapter V. ("Sensation") opens with a statement of the general law of the physiology of sense:—"Every sensory nerve fibre, however excited, brings a sensation to consciousness, which differs from any sensation that another nerve fibre can mediate." In order to distinguish this single-fibre sensation from the "sensation" in the sense of a presentation complex, it is termed a "sensation element." Even the sensation element has a comparatively complicated physiological substrate, as is shown by the fact that it possesses quality or modality (in virtue of which it is comparable with other sensations), a variable intensity, and, in certain cases, a local sign. Variation of intensity involves change of quality.[1] The writer rejects Helmholtz' definition of sensation in favour of its characterisation as "an unanalysable portion of a sense-impression, with no attributes beyond those of quality and intensity." "The description of a sensation is unintelligible except for those who have it in memory, if not for the moment in consciousness."

Where the law of isolated conduction from sensory nerve termination to cerebral cortex is valid, we may speak of "primary" sensations. But it is probable that the range of its validity is much smaller than is usually thought—if, indeed, it is really valid at all. Beside the primary sensations, then, we have "secondary," which "arise from the interaction of two or more excitations in nervous organs, simultaneous or successive." There is no conscious mark of secondariness. Secondary sensations are local (a hunter can just see the game in the twilight, it becomes invisible when he closes one eye to take aim); temporal (tickling of the lips in playing a trumpet); and both (sensations aroused by the movement of a perceived object). "Change as such excites a specific sensation."

The feelings are "sensations from internal organs, arising secondarily as the result partly of centripetal, partly of centrifugal

[1] This statement rests upon an objection to Fechner's law, urged in a different connection by Hering. It is that, if the law holds, "the increase of a 1cm. line to 10cm. must cause a sensation indistinguishable from that caused by the increase of a 1m. line to 10m., *i.e.*, 9cm. must appear to us as large as 9m." This is surely an erroneous interpretation.

excitations, and arriving at the organ of consciousness as do other sensations," *i.e.*, are a kind of concomitant sensations. They are ordinarily set up by "the release by a central or peripheral excitation of a subcortical central process, which either evokes muscle sensations directly or starts centrifugal stimuli, which travel to the muscles and cause the sensations of muscular contraction in the corresponding sensory nerves." The most important feelings are pleasantness and unpleasantness, each of which is independent, *e.g.*, of the degree of intensity of other simultaneously present sensations. They consist essentially (1) of sensations in the thoracic cavity, due to changes of circulation, and (2) of "muscle feelings." Mixed feelings occur, in consequence of the partial identity of the muscle groups concerned. Some nerve endings always respond to excitation by pleasure, others by pain. The pain following intensive excitation of a "sensory" nerve, in the ordinary sense, may be mediated by special pain fibres, or by the "sensory" fibres themselves. A diagram shows the functions and connections of the "pain centre," which is regarded not as a separate central organ, but as composed of "the higher nervous centres of all the muscle groups which are in any way concerned with the movements of defence. These may be situated in quite different parts of the central nervous system."

A concluding section treats of "combinations of movements and secondary sensations in their relation to inheritance." The author shows how the reflex may be derived from the voluntary action, and how the implied cell-relationship may be inherited, on either of the current theories of hereditary transmission. There is a continuous series from strictly secondary sensations to analysable complexes (violin clang); and from irresolvable combinations of movement to combinations which can be broken up. The mechanism of the less rigorous connections is given in diagrammatical form.

A perception (Chapter VI.) is "a unitary complex of excitations, which can be split up by consciousness into sensations." There is no absolute line of distinction between sensation and perception; the passage is by way of the analysable secondary sensations. Perception becomes idea by association with memorial images. This complex, which Helmholtz, *e.g.*, technically names "perception," Professor Exner calls "secondary perception," reserving the term "primary perception" for the perception of his original definition. The chapter falls into three sections. (1) *The excitations in the organ of consciousness.*—The cortex is com-

posed of nerve paths, in the widest sense of the phrase. It is a result of physiological thinking, to be verified *à posteriori*, that "all the qualitative and quantitative phenomena of conscious sensations, perceptions and ideas, are referable to quantitatively variable excitations of different portions of this sum of paths." Two things follow. "Two sensations are identical for consciousness, when the sense-stimulus excites the same cortical paths to the same extent." And "two sensations are similar, when at least a portion of the paths excited in the two cases are identical." Since the path of a sensation element divides at the centre, or between periphery and centre, "the quality of a sensation and its local sign are the result of the excitations of different paths within the cortex." These four propositions are fundamental. It may be inferred from the last that there is a "perception element," the sum of sensations due to the excitation of a single sensory fibre at the periphery; an element, *i.e.*, which has a highly complex physiological substrate. Not all the cortical excitations of the moment are represented in consciousness, but all may influence conscious content, and may be consciously apprehended (thus differing from the excitations underlying strictly secondary sensations). "These cortical excitations, which do not come independently to consciousness, play a large part in the psychical life." (The reader is reminded of Müller and Schumann's work on "Memory," or Külpe's on "Attention.") The phenomena illustrate a principle already employed by the writer in other connections, that of "central confluence." "A sum of excitations in the cortex, following different paths, can act upon consciousness as a single excitatory process." The cortex is never wholly unexcited. (2) *The Primary Perceptions.*—Like sensations, these are really abstractions. The primary perception "is the process which we must suppose to be present in the cortex of a newly-born child, or of a congenitally blind person, deprived also of tactual and muscular sensation, who has suddenly been given the power to see and feel." (3) *The Secondary Perceptions.*—Here we have to consider, in the first place, the "local impressions" which constitute, *e.g.*, the "consciousness of direction," so highly developed in many animals and some men. Ruling out visual sensations for the moment, we can analyse this consciousness into "obscure" perceptions and recollections (innervation and organic feelings, recollections of obscure perceptions of bodily attitude). "Processes run their course in the cortex as obscure perceptions, and their results are embodied in consciousness, without its being possible for the factors which enter into them to be apprehended by con-

sciousness after their occurrence." Local impressions lead us, secondly, to the "principle of recognition," of our capacity of distinguishing whether a cortical excitatory process is new or repeats an earlier one. Our recognition of the excitation of a path is explicable in terms of two physiological laws. The first is that of the "rutting a path" by usage—the setting up of a kind of permanent facilitation. The other is that of the arousal of secondary excitations. A bloody finger means the excitation of the rutted red-path. But this path was traversed previously by the redness-excitation of a cherry, and an "intercellular tetanus" effected between all the cherry-cells. The blood consequently excites (weakly) all the cherry-paths, by exciting (strongly) the red-path. These excitation complexes, which do not correspond to the condition of the subcortical centres (being due to attentional facilitation), are "memorial images." It is to be noted that a cortical excitation complex need not be for consciousness the sum of the separate excitations, though it will resemble that sum. (We may *cf.* Külpe's definition of sensation fusion.) Thirdly, we come to the eye, to the retinal "local signs." There are two constituents in local signature. "Muscle feelings" account for the "sensation complex, evoked by a visible object as a whole." To explain "how parts of an object or several different objects can be correctly cognised as regards their relative position with apparent simultaneity," it is necessary to call in the aid of "unitary secondary sensations, varying with the intensity of excitation of the particular paths which belong to them." Subcortical relations are developed between the central elements connected with neighbouring retinal elements, or with corresponding elements of the two retinæ. We must also assume a cortical relationship between the terminations of "similar," *i.e.*, neighbouring retinal elements. The discussion of the two factors in local signature is very elaborate. Reference is made to the development of the muscle feelings in the animal kingdom under the general law of teleology; and their action is demonstrated by a diagram, like that used to illustrate the visual "movement sensations" of Chapter V., 2. Another diagram shows the cortical network corresponding to the relations of the retinal elements. Pathological cases are adduced to support the theory that localisation occupies different parts of the cortex from those concerned in the other functions of vision. Localisation may be impaired alone; vision may be practically abolished, with the exception of the movement sensations; colour sensations may be absent, while those of brightness and locality are retained.

The "similarity" of sensations is examined, in the light of the law of specific function (Chapter V.). Lastly, the complication of the perceptions already spoken of by the perception of depth is indicated.

Chapter VII. treats of ideas, which differ from perceptions in two points. The idea is a perception *minus* certain excitations in the cortex—those which represent sensation in its purest form, as least "worked over" psychically, *i.e.*, those of the radiation of the sensory nerves. And the result of attentional facilitation differs in the two cases; in perception, it finds excitation in the subcortical centres, in idea it finds them at rest. Individual variations are more important in idea than in perception. The underlying excitation is diffused over a large portion of the cortex. There are "obscure ideas" as there are obscure perceptions, playing their part "outside or almost outside of consciousness."

The first section of the chapter raises the question of the nature of this (1) *consciousness*, of which so much has been said. "When an excitation-complex in my cortex has attained a certain diffusion . . . and has so come to involve the paths which have been more particularly excited by events of my own experience . . . and which are therefore almost always in a state of facilitation—the paths, in a word, which belong to my idea of myself; and when the excitation has increased in intensity by way of intercellular tetanus, owing to the number and variety of the excited fibres, and the excitation-complex has consequently acquired the . . . peculiarity of inhibiting weaker excitations; then I say that the idea is in consciousness." The consciousness "into" which the idea "enters" is a group of associated ideas. Self-consciousness "depends exclusively upon ideas of previous experiences, more or less distinctly associated to the ideas or perceptions present at the moment." The cortical fibres concerned in voluntary action (Chapter III.) form part of the complex whose excitation gives the idea of self. (2) *Ideas in their Relations to Voluntary Movements.*—Before acting, we have an idea, however transient, of the result of our acting. Having acted, we possess a perception, with which we can compare and by which we can check our idea. The mechanism of these processes is explicated. (3) *Ideas in their Relations to Sensations and Perceptions.*—Very different primary sensations can produce practically identical perceptions, because of the wealth of the cortex in associative "ideational material." On the other hand, this ideational material can facilitate or inhibit

sensations. The modificatory processes are probably cortical, as well as subcortical. Instances are given under both rubrics. (4) *The Interrelations of Ideas.*—In the last resort, all ideas originate in perceptions. We are able, however, to bring about relationships between cortical paths which have not been previously effected by perception. The limit of imagination is reached when the path-complexes excited are partly identical: we cannot ideate a "curved straight line" (identity of the motor mechanism). No idea can be retained for any great length of time. It fluctuates intensively, probably for vasomotor reasons. It also oscillates with other ideas, probably as the result of the rapid exhaustion of the cortical organs. (5) *The Arousal of Ideas by Words, and the General Effect of Sensational Succession.*—The fact that the spoken words "St. Paul's" arouse the total idea of the cathedral can only be explained by the consideration that these words constitute a succession of sounds. Other words contain similar separate sounds ("pall," "awe"); but as the two are uttered, there is a progressive restriction and definitising of the cortical paths excited. And this holds of single words. The most primitive languages employ sounds as such; the more developed, sound-successions. Although we have in adult life no consciousness of understanding words which we hear in terms of their sequence of letters, this is only because of the extreme rapidity of the process of "successive selection." Sequence-understanding is rendered possible by the persistence of nervous excitations after the cessation of stimulus.[1] It is true that the motor innervations of the listener play some part in his understanding, but they cannot be the sole factor. The idea of the word "St. Paul's" is, of course, different from the idea of "St. Paul's." It has visual, motor and auditory constituents. The two ideas are closely associated. The auditory word idea is of brief duration, and can only be revived by aid of the motor.

We have now reached (Chapter VIII.) the most complex mental processes—the phenomena of intelligence. (1) *Forms of Thought.*—The formation of a *judgment* can take place in two ways: we judge, when a difference between ideas, given as different, is apprehended (*erfasst*) by consciousness; or when we analyse out, by attention, the constituent parts of the excitation-complex corresponding to a single idea. If we direct consciousness upon the cortical processes common to a number

[1] It must be remembered that the memorial images of the separate sensations differ not only in intensity (according to their temporal order), but also in quality (*cf.* Chapter V.).

of ideas, we have a *concept*. Judgment and concept are thus not necessarily connected with language, though language is undoubtedly of service for both—more important for the concept than for the judgment. *Inference* (*cf.* J. S. Mill and Helmholtz) is a "form of words, in which is expressed a judgment together with the circumstances which led to its formation." *Reflection* always involves "an intensive attentional excitation of all the cortical fibres constituting the idea which is the object of reflection, and the consequent facilitation of related fibres, near and remote." This given, thought may follow either of two roads. We may have a "happy thought"—a new association being formed between ideas hitherto separated—or we may "experiment" by facilitating all manner of related paths, and looking out for contradictions in the excited ideas. Serious thinking ordinarily depends upon a combination of these processes. A *resolution* is a judgment passed after experimental reflection, and is always on the side of the ideas which are associated with the maximum of pleasure or the minimum of unpleasantness. These feelings are represented in the cortex directly, but also indirectly (reaction from sub-cortical centres). It must be remembered that the part played by the will in the processes of reflective thought and resolve need be but very small. "Thought goes on in me" is a truer expression of the facts than the customary "I think." (2) *Instinctive Feelings and Thought.* —It is more than possible that "a connection of a cortical complex of a definite kind with the centrifugal fibres running to the feeling centres will ultimately take its place among the transmissible nerve paths, provided that it has subserved an important physiological function for many thousands of years." Ideas, it is true, are not inherited; but there may be transmitted a disposition, an inclination towards the colligation of certain not sharply defined ideational masses with determinate feelings. The feelings are "measures of defence" taken for us by nature. The associations of feeling and idea of which we are speaking result in the protection of the individual, the benefit of his immediate descendants, or the benefit of society. The organic faculty of unconscious action for a definite end we call "instinct." "An experience which runs counter to our instincts is painful, and there is no other means of causing pain." Hence the excitations in the centres for pleasure and unpleasantness may be termed "instinctive feelings." These feelings are discussed at length under the three headings just mentioned—their discussion taking the author some distance into the domain of ethics and sociology.

(3) *Causal Thinking and Free Will.*—"There are congenital relationships in the human cortex, as a consequence of which the sensation of a change (which, of itself, attracts the attention, and, coming to consciousness as perception, affects a portion of the motor cortical area) excites further, and more especially motor paths subserving the search and effort required for the investigation of the change, *i.e.*, the obtaining of associations for it. So far as regards the change, the only associations that can be found are 'causes.' This congenital endeavour after the associations of the change we term the law of causal thinking." Since ideas are not inherited, it is an inclination only, not a constraint, which is congenital. There are biological reasons for the importance of the perception of change. It is curious that although we cannot conceive of effect without cause, most of us actually recognise uncaused effects. While we are dealing with the external world, it is plain sailing with the law of causation. The objective psychology of the present book, *e.g.*, is causal. When we speak as experiencing selves, however, we cannot run the causal chain further back than consciousness; we know nothing of the unconscious physiological processes posited by the man of science. Hence, to the naive mind, "free will" is the last cause of action, the uncaused effect with which the causal nexus begins.

Two remarks seem called for by way of corollaries. (*a*) We cannot clearly ideate a process without cause. Hence the causal law is a psychical phenomenon. But we think that we act by free will; this is also a psychical phenomenon. We need not strive to reconcile the contradiction. Causation may be the most useful guide in one sphere (scientific thought); free will in another (individual and social education). That the causal law is built into the physical structure of man, in the manner described, makes no difference to this conclusion. (*b*) We have spoken of free will; we have not yet said what "will" is (cf. Chapter III.). It is nothing *sui generis*. When the cortical cells are sufficiently charged, *e.g.*, after sleep, some stimulus excites a group of paths, and intercellular tetanus—the play of ideas—begins, whether we will or no. "There commences the normal interplay of associations and (where the excitations are transferred to motor cortical fibres) of actions, which are performed in accordance with the associations, and present themselves to the subject as voluntary actions." Their energy is due to the charge of the cells (sleep) and to all the associative excitations in the cortex (also intensified by sleep). At the close of the day, the "energy of the will" is lowered; action becomes weaker.

The index is scanty, and not always correct.

There can be no doubt that Professor Exner has worked out a plausible and consistent theory. This is not to be wondered at, when we remember that the *Entwurf* is the result (as I have said at the outset of the present notice) of twenty-five years' occupation with physiology, "during which the attempt at an explanation of psychical phenomena came to be more and more definitely regarded as a life-work." The greater part of the author's published writings sprang, as he tells us, from this one interest. I have aimed, in the above summary, at bringing out the psychology of the volume, that the reader may be able to compare the conclusions reached by purely physiological methods with the outcome of strictly psychological analysis as given in the best modern text-books. The summary is so brief that very many points of detail, important in themselves, have been passed over; while it is of the nature of a summary to emphasise the breaks in the argument rather than its continuity. Moreover, it has been impossible even to mention the light-giving personal experiences with which the discussion is interspersed. And nothing has been said, beyond the barest statement of their existence, of the nervous schemata which form a large feature of the work, and constitute an integral part of the writer's "explanation."

As a constructive synopsis of the scattered results of physiological investigation of the central nervous system, and as the first thoroughgoing attempt at the realisation of an "internal psychophysics,"[1] the *Entwurf* will be extremely valuable both to physiologists and psychologists. Under either aspect, of course, the book has the defects of its qualities. We see the weak places in the methodological framework which holds the physiological data together; and we see, I think, that current physiological

[1] This characterisation may seem to conflict with what was said above of Wundt and "physiological psychology." But a comparison of the *Grundzüge* with the *Entwurf* will show what is meant. Wundt devotes 150 pp. and three chapters to anatomy ("structural elements of the nervous system," "morphological development of the nerve centres," and "course of the paths of nervous conduction"): Exner 30 pp. only. Wundt's two physiological chapters ("physiological functions of the central parts," "physiological mechanics of the nervous substance") and Exner's one (*die physiologischen Grundphänomene*) are of approximately the same length: but what in Wundt is a summary to be referred to for "explanation" in later chapters, is in Exner introductory to an elaborate series of very detailed explanations, which practically constitute a continuation of it. The problem is essentially the same; the way of approaching the problem is different. Wundt connects a minutely subdivided mind with a generalised substrate: Exner a minutely analysed substrate with a somewhat roughhewn mind. It is the definitising of the excitation-process, however, which is the keynote of "internal psychophysics."

concepts are incapable of furnishing a really adequate "explanation" of mentality. That, at least, is the impression left by the work regarded as a whole; and the impression has been confirmed by a detailed comparison of psychical facts and alleged physiological conditions in two or three special cases chosen somewhat at random. Professor von Kries has worded this judgment more forcibly from the physiological point of view than I should have ventured to do from the psychological. "I cannot get rid of the thought," he says, "that psychology presents a whole series of problems for which current physiological ideas do not furnish any adequate expression. . . . These considerations have kept me in doubt as to whether the physiology of the central nervous system, and more especially of the cerebral cortex, must not come in the future to work with quite different ideas from those which it now employs (excitatory process and its conduction)."

E. B. T.

Various Forms of Hysterical, or Functional, Paralysis. By H. Charlton Bastian, M.A., M.D., F.R.S. Lewis, London, 1893. 1 vol., 8vo., pp. 197.

Dr. Bastian's work is well timed and of great interest. He applies to the study of hysteria a method formerly almost exclusively employed, but of late years—perhaps erroneously—somewhat neglected. We mean the *anatomo-pathological* method, which has given such brilliant results in the study of nervous diseases, and which had been also attempted in the interpretation of hysterical phenomena. Thus our respected and regretted master, Professor Charcot, compared,[1] in his early work, hysterical hemi-anæsthesia with that observed in brain lesions, especially in those of the posterior part of the internal capsule.[2] Dr. Paul Richer did the same in a memoir (1882) recently published in a work on hysterical palsies and contractures (1892), when he described symptoms due to disordered cerebral innervation, and paralyses, "the dynamic lesion of which must be sought in the inferior part of the cerebro-spinal axis." Bastian thinks that this method has not yet given all its

[1] Charcot, "Leçons sur les Maladies du Système Nerveux," i., 306.
[2] *Ibid.*, iv., 114.

promised results, and that it must be applied again and more rigorously. His book is an interesting and enterprising effort: it is an anatomical study of hysteria.

Anatomo-pathological studies are usually done by dissection, microscopical examination of organs, and minute comparison of observed lesions with symptoms observed during life. Unfortunately, this procedure, rarely applicable in hysteria, has hitherto failed to yield the least result in the study of the disease; and on this point the work before us does not differ from the others. Though an anatomical book, it does not contain a single figure. The author is obliged to have resort to the indirect and much more dangerous method, already employed by Charcot—the method of *comparison*. Hysterical symptoms, with unknown physical substratum, are compared with those of other more or less analogous diseases, but with known lesions, and conclusions are drawn from the ones to the others. Thus contractures, or some contractures, in hysteria are assimilated with hemiplegic spasms through descending sclerosis of the pyramidal tract; some hysterical paralyses are compared with atrophic infantile palsy through anterior cornual lesions; and one concludes that the hysterical contracture depends upon a lesion of the pyramidal tract, the paralysis upon a lesion of the anterior horns. This mode of argument, which pervades the whole book, arises from the author's conception of the so-called functional diseases as compared with the organic, or lesional, diseases. We must examine this point more closely.

Neuroses, such as hysteria, of which the lesions escape our eyes, are often considered as functional or dynamic disorders, as opposed to the organic disorders with known lesional substratum. These very vague expressions merely serve to hide our ignorance. Bastian very rightly tries to give precision to this idea. He does not look upon the two as so distinct as is usually done; and he shows well how in some cases a diagnosis is difficult, and that hysteria can show incontinence of urine, atrophy, even eschars (very rarely, we think), which had been considered as characteristic of organic diseases (pp. 85, 117, 123). He insists, with several recent writers, upon the frequent association of hysteria with organic lesions, such as insular sclerosis—Pott's disease (pp. 67, 72). He even thinks that in some cases a long continued functional paralysis may be transformed into one with actual lesions. Bastian recalls an old saying of Charcot's,[1] that a long continued hysterical contracture may bring about a sclerosis of the corresponding pyramidal tract. We think, how-

[1] *Ibid.*, i., 362.

ever, that as a natural consequence of his prolonged researches, Charcot would not have latterly accepted, without reserve, this opinion; and it is a pity that our author should not have fortified this old view by fresh proofs. Bastian concludes that there is a great analogy between the two forms of nervous diseases, which really differ in three points only: (1) Lesions have not been observed in functional disorders; (2) Their symptoms are merely attenuated, reduced. "Looking at the matter from another and, I venture to think, more rational point of view, we may see that the phenomena of mere functional disease should not be expected to differ in kind, but merely in degree, from the forms of structural disease to which they are akin" (p. 122). (3) Functional diseases are chiefly characterised by their lesser duration, by their recovery. In a word, the lesions in functional troubles are slight, transitory, due probably to some vaso-motor spasm (pp. 7, 45), but they occupy the same regions and provoke the same symptoms by the same mechanism, as organic diseases.

It is not strange, therefore, to see hysteria simulate brain or cord affections, since the symptoms are fundamentally the same, and arise from defects in the same substratum: "It simply comes to this, that the symptomatology of different forms of paralysis is governed in the main by the part of the nervous system which is at fault" (p. 122). In the study of these affections we must return to the essential point, the *localisation*; their functional character does not dispense us with seeking their exact cerebral or spinal seat (p. 2). We must be able to distinguish a functional defect of the cerebral cortex of the lumbar cord (p. 5). Thus shall we be able to make distinctions in this over-loaded group of functional diseases, and recognise the fact that many differ from others, and must not be designated by the vague term "hysterical."

We have endeavoured to summarise correctly the author's view on this essential point; we may now, without criticising it, indicate wherein the further study of the book leaves us still in difficulty and hesitation. The differences which Bastian allows to subsist between organic and functional diseases are after all of small import. The absence of observable lesions is not definitive, and depends on the insufficiency of our means of investigation. The duration and final cure of the disorder are not signs of any precision; hysterical symptoms may last for years and never disappear (an old Salpêtrière patient, Aurel, is hemi-anæsthetic at 75

years of age as she was at 20); whilst certain organic disorders (scleroses, compressions, peripheral neuritëdes especially, of which the author does not always seem to hold sufficient account) may be recovered from more or less completely. The third characteristic difference of intensity is not in itself of any value, and is furthermore not very exact. Hysterical symptoms, far from being always attenuated, are mostly very clear and intense. Hysteria, as Charcot said, is essentially a disease with "excessive manifestations." Its palsies, anæsthesiæ and contractures are more definite and pronounced than elsewhere. Thus the differentiæ between organic and functional diseases tend to disappear. Perhaps the two orders may still be etiologically distinguished; but clinically they will run into one another and consider their localisation only. The group of hysterical symptoms should disappear as useless. But may we accept these conclusions; and are hysterical contractures and palsies clinically undistinguishable from organic nerve symptoms? And have the words "functional troubles" lost every meaning? The expression "functional disease" appears to us as possessing some exactness even when taken in its literal sense; it is a disease of the "function" as opposed to the organ. The function is an *ensemble* of acts executed most frequently by several organs, co-ordinated so as to work out a common end. It is rare that an organ, taken singly, accomplishes a function; it mostly collaborates with other organs to fulfil a single function; and inversely it often enters as a constituent in several functions. The leg is an organ, and walking is a function; the legs are associated together and with the muscles of the trunk in carrying on the function; and on the other hand the leg comes into play in other functions, such as dancing, jumping, &c. An affection of the leg leaves the other parts intact but suppresses its action in all functions, dancing as well as walking. On the contrary, a disease which allows the patient to jump, to dance, move the legs in bed, and push them firmly against an obstacle, but which makes walking, and walking alone, impossible, is a disease of the function. Disturbances thus systematised are frequent; thus, in astasia, the patient without paralysis of a single muscle cannot stand; in abasia he falls as soon as he tries to walk normally, whilst he can skip over a rope or go on tip-toe. The same obtains in the numerous professional neuroses, in writing, sewing, &c., when the hand is fully capable to accomplish all other movements; in mutism, with loss of speech alone without any interference with the intellectual processes; still more in the loss of one's native language, with

preserved use of a foreign tongue, &c. In such very definite cases the disturbance of function stands in strong contrast with disease of an organ.

But, will it be said, when the whole limb is paralysed the distinction is more difficult. No doubt, but the characteristic systematisation still exists. The paralysis is too complete, and is too sharply limited to be explained by coarse anatomical lesions. The hemiplegia affects the arm and the leg, without invading the facial area; it is a "double monoplegia" rather than a hemiplegia as Charcot frequently puts it. The arm is completely paralysed, and total anæsthesia extends upwards to the shoulder like a coat-sleeve. The eye is wholly anæsthetic, including lids and conjunctiva; and yet there is not the least implication of the facial or the leg, and the rest of the face preserves its complete sensitivity. It would seem that the function of the organ, and more especially the natural and common conception people have of this function, plays a more important part in the localisation of the symptoms than the anatomical arrangements of the limbs and nervous centres.[1]

It may be said that this very systematisation, this function of walking, writing, speaking, must have its anatomical counterpart, its unit in some single cerebral organ. It is possible; but there is absolutely no proof of the fact. The distribution of nerves and arteries, the cerebral centres, represent coarser systematisations, and determine movements of organs participating in various functions. We know in a way the centre of lip-movements; but what about the centres presiding over the articulation of a French, English, or German word? Hence the saying: "Hysteria behaves in its paralytical and other manifestations, as if anatomy did not exist."[2] Far from being explicable through our knowledge of cerebral localisations, hysteria is opposed to our *actual* knowledge of brain mechanisms.

The term "functional disease" may be of a temporary description, which will become useless when we shall have accurately localised all these complex and delicate systematisations; but until then it has a meaning and designates a set of phenomena different from those named "organic."

These remarks will find their application when we follow Bastian in his anatomical classification of "functional" disturb-

[1] We have often endeavoured to put this point in evidence (see especially our lecture on "Hysterical Anæsthesia," *Arch. de Neurol.*, 1892, p. 329. See also Freud, *Ibid.*, 1893, p. 29).

[2] Freud, *loc. cit.*

ances. He distinguishes a certain number of paralyses, which he connects with cerebral troubles; and these he considers as hysterical properly so-called. What justifies, to his eyes, this localisation is the single—important enough—characteristic of loss of muscular sense, or kinæsthetic anæsthesia, which accompanies these paralyses. Hence the author places their seat in the Rolandic and marginal convolutions which govern the affected limbs. When these symptoms are complicated by sensory anæsthesia, a similar defect must exist, according to Bastian, in the sensory portion of the internal capsule, the portion of the corona radiata which is between the optic thalamus and the posterior portion of the lenticular nucleus (pp. 18, 121). Finally, in certain cases of hysterical mutism, or complete simple aphemia with intellect unimpaired, the author assumes a disturbance among the descending fibres from the Rolandic convolutions, and the associative fibres which connect the kinæsthetic speech centre of the third frontal sulcus with the bulbar centres of articulation (p. 50).

We must pass over the two last localisations, the full discussion of which would lead us into the wide field of hysterical anæsthesia and mutism; a study of the first and most important will, moreover, cover some of the ground. Rey Régis ("Histoire Naturelle de l'âme," 1789) had already recognised the existence of motor paralyses, depending on loss of memory of motor force, due to certain cerebral lesions; and Charcot had also mentioned this forgetfulness of the motor images.[1]

Bastian endeavours to give precision to these ideas. He recalls his own important theories, in which he established the sensori-motor function of the Rolandic area. These centres register kinæsthetic impressions, preserve the images of the muscular sensations felt in the performing of the several movements, and by the reproduction of these images determine and guide the reproduction of the movements. Destruction of functional disturbance of these centres must give rise to three symptoms: (1) loss of muscular sense in the corresponding peripheral organ, loss of sense of position of the limb, of sense of weight, &c.; (2) loss of images related to the movements of the organ; (3) its motor paralysis. Horsley's cases and experiments show this to be the case for destructive lesions of the ascending convolutions. Bastian assumes that similar conditions obtain in hysterical paralysis; the affected limbs are paralysed as to movement and

[1] *Loc. cit.*, ii. 417, iii. 464.

muscular sense. The author notes this curious fact,[1] that the patient cannot recall to his imagination the movements of the affected limb: "Again, when asked whether she can imagine herself playing on the piano with the left hand, she at once says 'yes,' but in regard to the right hand she gives the same negative reply as before" (p. 15). This functional inertion of the Rolandic area may be more or less complete; at a lower degree it produces a slight disturbance of motion, and may, for instance, cause the curious disorder which we have studied[2] under the name of "Syndrome de Lasègue." The patient moves normally with the eyes open, but cannot move the anæsthetic arm when they are closed (p. 27, 213). When the cerebral inertia is more intense it brings about true paralysis; thus the assumption of a functional disturbance of a localised brain area explains all the phenomena of hysterical paralysis.

We confess that this explanation appears to us rather too simple to account for such delicate and complex phenomena. No doubt it contains a portion of truth, and the Rolandic kinæsthetic centres may count for something in the production of hysterical paralysis; but to explain all by defects in them, it is on the one hand to say too little, on the other to say too much.

To speak only of insufficiency of the sensori-motor centres is to say too little, for hysterical paralysis is a highly complex phenomenon of which amnesia of kinæsthetic images is only one element, and often a secondary element. That a Rolandic centre be exhausted, that may be; but why should it remain exhausted a whole year? What is it that prevents reparation and keeps up this persistent exhaustion? Why should exhaustion affect the particular organ, and follow on a certain event, whilst it does not follow on some other much graver occurrence? In reality many other phenomena accompany this one, such as associations of ideas, curious troubles of attention, which play an important part in Lasègue's "Syndrome," and especially dreams, conscious or unconscious, fixed ideas, which play a capital part in the accident, explain its duration, its localisation and systematisation. It is not without reason that Brodie, Russell Reynolds and Charcot have spoken of paralyses "dependent on idea." Is it possible to discuss these phenomena without speaking of the mental states on which they depend? Bastian alludes

[1] *Cf.* P. Janet, "Automatisme psychologique," 1889, 347-362. "Etat mental des hystériques, v. ii., p. 117.

[2] P. Janet, "Automatisme psychologique," p. 350. "Stigmates mentaux des hystériques," 1892, p. 174.

too briefly to suggested paralyses; he does not notice that in these paralyses, the exhaustion of the Rolandic centres, if present, is quite secondary, and that everything depends on the suggested idea. Is there any thorough-going difference between experimentally suggested and naturally developed paralyses? If their nature be identical, as many observers have affirmed, the exhaustion of the muscular centres is in neither category the essential factor. We shall relate a curious case, the last we had the opportunity of studying under Charcot. A young man, L., notices one morning on waking up, a weakness of the right arm, which becomes more marked every succeeding morning, until it becomes an almost complete paralysis. The arm presents a complete loss of muscular and tactile sensibility; a patch of anæsthesia covers the anterior and lateral aspects of the neck; and a painful and hyperæsthetic spot is found on the left flank. Shall we say that there is only exhaustion of the foot of the second frontal convolution, and another curiously limited one in the sensory part of the internal capsule? Is it not more just and scientific to take into account the facts elicited by an examination of the mental state of the subject? This young man has dreams which he expresses aloud during natural sleep, somnambulism, and attacks provoked by pressure of the hyperæsthetic point. He is a great reader of sensational novels, and has had a dream connected with the fact that his window opens on a balcony; he dreamt that a robber entered into his room and attacked him, by squeezing his neck with one hand and pressing his left flank with the knee; whilst he himself was repelling the aggressor with his outstretched right arm. The patient reproduces the scene during his attacks, when with the right arm, *no longer paralysed*, he tries to keep off the imaginary foe. Are not the symptoms localised in the neck, in the arm, in the left flank well explained by the dream? The cutaneous sensations of the neck, the feeling and movements of the arm involved by the dream, are, as it were, absorbed by it. Since the dream persists, as we have often verified it, during the waking state, though unaware of the subject, these phenomena are removed from the cognizance of the conscious personality, and their absence determines anæsthesia and paralysis. From this simple and typical instance it will appear that it is not enough to speak of insufficiency of the Rolandic area.

But it is also an exaggeration, as we said before, to speak of these centres as exhausted and impotent; it is to say too much. The gravest paralysis may disappear for a time under particular circumstances. We have elsewhere shown that the symptoms

may disappear during attacks, delirium, intoxication, normal sleep, and especially during somnambulism.[1] "The physical exhaustion must be very slight to disappear so suddenly during the course of the malady, and the influences capable of modifying it so, appears to be the more important factors." But there is a most important topic on which we have insisted for years.[2] We have shown that in the deepest hysterical anæsthesia and paralysis neither sensation nor movement was completely abolished. They subsist and often can be brought into evidence by simple processes; but they subsist unaware to the subject, in a sub-conscious form, without his being able to perceive them, that is, connect them with his own personality. Things take place as if the elementary psychological phenomena were as real, as numerous as in normal subjects, but could not, on account of the shrinkage of the field of consciousness, and the weakness of synthetic power, cluster into a single perception of personal consciousness. We need not insist upon the theories by which we have tried to account for very complex facts; but our experiments, verified by others (*e.g.*, James, in America; Pick, Breuer, and Freud, in Germany; Binet and Laurent in France), show that we must not hastily assume arrest, exhaustion, functional insufficiency of the cortical centres in these hysterical phenomena. We cannot, therefore, accept Bastian's localisations as sufficient to account for all the facts of cerebral hysteria.

The author now comes to a very different subject. He thinks that certain functional paralyses, apparently hysterical, are so distinct from those we have been discussing as to require a different explanation. They depend upon spinal insufficiencies. This conception of spinal diseases, analogous to hysteria, inasmuch as they are functional yet very distinct as to their real and true nature (pp. 12, 55, 119), is certainly bold and interesting. It has already been broached by other authors (P. Richer, &c.), and may contain the germ of an important nosographical classification. Hence we must examine it carefully, and weigh the reasons adduced by the author in support of the distinction between these medullary paralyses from cerebral hysteria. Bastian describes two forms, the one accompanied by contracture due to pyramidal disturbance (57), the other flabby, due to diminished

[1] P. Janet, "Les accidents mentaux des hystériques," p. 121.

[2] P. Janet, "Les actes inconscients et le dédoublement de la personnalité, *Revue philos.*, 1886, vol. ii., p. 577. "L'anesthesie systématisée" and "La dissociation des phénomènes psychologiques." *Ibid.*, 1887, vol. i., p. 449. "Les actes incomscients et la mémoire pendant le somnambulisme," *Ibid.*, vol. i., p. 238.

activity of the anterior cornual cells, and which may even be accompanied, in advanced cases, with some atrophy (97). The author, it is true, readily admits the somewhat adventurous nature of the suggestion; and we need only dwell upon the essential point, the attribution to the spinal cord of certain paralyses admittedly functional and unexplainable by any of the known types of anatomical alteration of the cord or nerves. The author's reasons for establishing this distinction are found somewhat disseminated through his work. He often insists upon the mental peculiarities of the patients. "During the whole time of her stay in the hospital she had never manifested any undue emotional disturbance. Her mental condition has been fairly equable, and there has been no tendency to exaggerate her troubles or disabilities" (p. 65). Similar remarks occur frequently (pp. 79, 90, 101); and yet reduced to these terms, can such observations be decisive? No one to-day holds to the old legend of the ever changeful, gay or amorous hysteric; among men and women too, totally different moods have been found to exist. The apathy noted more than once (pp. 75, 102) and opposed by Bastian to the hysterical temperament, we do not know why, is quite consistent with it. But we need not insist; in order to prove anything in the matter such observations must be much more precise and full.

Bastian also mentions the absence of attacks. This is more to the point; but have those patients never had any of the numerous equivalents of attacks? The author mentions mutism (p. 92), which should be carefully investigated, and do not point to a medullary trouble. Moreover, we know that attacks are often missed, especially in cases analogous to those adduced. When a hysteric suffers from a permanent symptom, contracture or paralysis, it frequently happens that no other symptom is developed, and that the patient is absorbed in her (conscious or unconscious) idea, and becomes unsusceptible to any other.

Bastian adduces contracture as a spinal, and not cerebral, symptom; as evidently similar to that which constitutes the first degree of spastic paralysis due to lateral sclerosis. This assimilation appears strange: the rapidity and exaggeration of hysterical contracture, its character when examined unknown to the patient whilst her attention is drawn away, its extraordinary modifications under anæsthetics, in somnambulism, its close relationships with the patients' thoughts and suggestions; all make it very unlike a spinal contracture. This contracture, as we have shown elsewhere,[1] is much more intimately connected with

[1] "Stigmates mentaux," p. 196; "Accidents mentaux," p. 109.

cataleptic and paralytic phenomena of cerebral origin. We do not wish to reject Bastian's hypothesis, which may be true in some cases, and we know that hysterical contracture is one of the most variable phenomena, difficult to analyse, but we may well ask for a more thorough examination of the facts before committing ourselves to such a grave conclusion.

The author next touches upon a very delicate topic, the examination of the reflexes, of the knee-jerk in particular, from the exaggeration of which he concludes to a spinal disturbance (pp. 58, 104). This exaggeration, also mentioned by others, was recently tested by us, by an examination of all the hysterical cases in the Salpêtrière. We noted that such an investigation is extremely difficult and requires the utmost precautions; and that owing to these difficulties and the enormous influence of the least emotion, the knee-jerk varies greatly even in the same patient. Even in paraplegics it was found to be irregular, *wild* rather than regularly exaggerated as in spinal affections. But should exaggeration be proved, it is open to discussion whether the fact does not depend upon conditions other than of purely spinal origin.

Bastian also mentions the absence of anæsthesia (pp. 101, 112); but we know that this is the case with many hysterics, and that typical anæsthesia is not common. Moreover, we have elsewhere shown that these patients without sensory disturbance have somewhat peculiar mental characteristics. We shall dwell more particularly upon a very important statement: that the patients did not show any sign of disturbance of the muscular sense in the contractured limbs. It is very difficult to test kinæsthesia under such conditions, and it is not sufficient to note that the patient has, with closed eyes, a knowledge of the position given to the limbs by the contracture (p. 62). For it is precisely a characteristic feature, that such patients have a persistent idea of the position of the limb, and cannot modify this idea. But when the contracture is incomplete, the presence of kinæsthesia can be made out, and Bastian justly lays stress upon the point:—"I take the view that it is of spinal rather than of cerebral type, mainly because of the absence of any distinct impairment of muscular sense in the legs, which, with such an amount of paralysis as exists here, ought to have been marked if the paralysis had been due to a functional defect in the Rolandic convolutions in relation with leg movements in the two hemispheres" (pp. 69, 96, 107). We think, if we may be allowed to say so, that the author is perhaps right, but does not convince us because he draws his conclusions rather precipitately.

We must examine—(1) Whether a paralysis without loss of kinæsthetic sense can be of cerebral origin; (2) Whether it is of hysterical nature? The answer to the first question will be clear if we consider paralyses through suggestion. In some cases they are accompanied with muscular anæsthesia, but very often occur without any. It depends upon the suggestion, the subjects' ideas, his previous psychological state, &c. Charcot had already noted the fact.[1] Bastian says as much, but does not appear to draw the due conclusions from the phenomenon, which shows that a paralysis without loss of kinæsthetic sensibility can be of cerebral origin. Moreover, clinical evidence is in accord with this statement; many paralyses clearly dependent on idea, especially if incomplete, do not involve the muscular sense. This obtains especially in astasia-abasia, and other similar systematised derangements. The paralysis is inhibitory, not amnæsic; there is a different mechanism, though evidently cerebro-psychological. As to the second question, Bastian remarks that all functional disturbances are not hysterical, and that paralysis without anæsthesia is not of this nature (p. 121). This involves minute considerations on the nature and limits of hysteria; and without entering upon them, we should not be indisposed to accept this view. Conscious fixed ideas, these pareses without muscular anæsthesia, may without inconvenience be separated from hysteria, which will thus remain more distinct; but it is not necessary to ascribe a spinal seat to them; it is enough to distinguish their different psychical characteristics.

The author has studied a number of interesting morbid states, difficult of classification; he wishes to create for them a special group, of functional spinal diseases. He may be right, after all; and his type may eventually become very important. But though the idea may be correct, the demonstration is not convincing; and for the present we are doubtful whether the cases cannot be reduced to one of the existing classes, that of organic lesions (pressure, neuritis, &c.), or that of functional cerebral troubles (hysteria, or some other psychical disturbance).

The author's bold attempt to give an anatomical account of hysteria is perhaps a little premature. At a time when we are scarcely beginning to understand the complex psychological mechanism of these symptoms, when we hardly know the coarse localisations of the simplest mental processes, may we hope to determine, with scientific precision, the seat of disturbed at-

[1] *Ibid.*, iii., 393.

tention, of dreams, of fixed ideas, of alterations of personality which play the chief part in hysteria?

The volume concludes with two essays on "The Neural Processes underlying Attention and Volition" and on "The Muscular Sense: its Nature and Cortical Localisation." These valuable contributions are known to the readers of BRAIN (April, 1887, and 1892). The monograph on the kinæsthetic sense is one of the clearest and best ever published, as befits the observer, who was one of the first to dwell on the importance of kinæsthetic sensations and images, and constructed the now classical theory on the subject. It is because we have all along studied with so much interest and profit the author's work on this subject, that we have ventured to criticise freely, though respectfully, this last contribution of his pen.

PIERRE JANET, M.D., Paris.

The Neuroses of Development. By T. S. CLOUSTON, M.D., F.R.C.P.E. Being the Morison Lectures for 1890. Edinburgh and London; 1 vol., 8vo, pp. 138, illustrated.

THE author presents an original and interesting picture of the various neuroses from a somewhat novel point of view. Instead of studying a neurosis, such as asthma or epilepsy, taken singly and in itself, he deals with these disorders in their mutual relationships, and in their succession in the same person at different periods of his life. Neuroses are presented as diseases of development—as troubles occurring in the nervous system at different periods of its evolution. In order to understand these accidents it is necessary to remember the various phases of this evolution in man. The author admits that the brain becomes adult, can exercise its full and normal functions at the age of 25 only; and he divides into two periods the period preceding maturity. During the first, extending from birth to about 18 or 19 years of age, the brain grows in size and weight. During the first seven years this increase is very rapid, and then becomes much slower. Some simple organs, such as the kidney, when they have reached their full growth, at once exercise their functions in a perfect manner. Not so with the more complex structures, such as the brain, in which the perfecting of the functions, after the organ has reached its full size, is very evi-

dent during a further period of about seven years. Without enlarging, the organ undergoes a developmental change, becomes more highly organised, and becomes capable of higher manifestations. This further organisation escapes our methods of investigation, and we fail to discover any appreciable differences between the brain texture of a child of 10 and that of a man of 25. That differences exist, however, is shown by the comparison of mind and conduct.

To this first division we must add another from another point of view, though no less important; the author calls "pre-reproductive," the period antecedent to puberty, which is followed by the reproductive period with its characteristic peculiarities. Hence the brain, in the pre-maturity period, goes through several stages, marked by critical phases: crisis of birth, ages of suckling, of dentition, of fastest increase of brain weight (4 to 7), puberty, period of greatest proportional increase in general body bulk, height, and weight next to the first year of life (14 to 17), of the gradual and steady maturity and solidification of the bones and tissues generally (18 to 25), and finally that of the completion of the organism, structurally and functionally, sexually and mentally, about 25.

These critical phases through which the brain passes in the course of its development are not without their dangers, and it is then that all original debilities mostly tend to become manifest. The latter depend on hereditary taint, and which is always found among the ascendants of epileptics and choreics. The author's volume is devoted partly to the study of these striking examples of degenerescence. He expands the theories of Morel and Moreau de Tours, insisting upon the fact that hereditary influences modify chiefly the organs whose development is slowest, and that these alterations manifest themselves chiefly at the time when growth and development have reached their summit.

The author, in common with Magnan and his school, endeavours to determine what are the apparent external manifestations of degenerescence, and one lecture is devoted to the description of some of the morphological signs of a bad neurotic heredity that appear during development. In several interesting passages he shows that physical ugliness must not be considered as an entirely normal fact, but is a sign of a faulty nervous predisposition. Thus in the case of J. R., he says (p. 21): "Her trophic centres have produced a coarse skin, a bad expression, a deformed and ugly person." And on page 34 we read: "There has been a lack of harmonious progress; the bodily ideal of the

age of adolescence has not been attained through the working of an evil heredity. Some features have been retarded in growth, some distorted, some overgrown." Many other corporeal defects are attributable to the same cause: asymmetry, dwarfishness, hunchback, squint, ungainly movements, horse-laughing, hobble-de-hoyism, uncomfortable limbs, malformation of ears, jaws, teeth, hands, and feet. Some of the author's statements are somewhat doubtful; at any rate, we may ask for proof that all these deformities really point to a neuropathic heredity.

The question of deformation of the palate and superior maxillary bone (ogival palate of the French) is more fully discussed. The author has made a special examination of the palate in numerous normal and abnormal subjects, and makes three classes: the normal palate, wide and not high; the neurotic palate, narrow and high (V or saddle shaped); the irregular and deformed palate. Statistics show that the latter forms are much more frequent among idiots and degenerates; and they correspond with a narrow cranial base—in other words, with a narrow brain, especially in the anterior lobes.

Persons offering such stigmata of degenerescence are subject to various nervous diseases, which manifest themselves at various periods of development. Morbid heredity is the stronger the earlier it becomes apparent, and may even disturb the embryonal functions, producing acephalous, harelip, cleft palate, spina bifida, deformed palate, and, above all, genetous idiocy. Later, during the period of greatest brain growth (up to 7 or 8 years), degenerescence is marked by rickets, night-terrors, convulsions, infantile paralysis, epilepsy, stammering, hypermetropia, convergent strabismus, lamellar cataract, choroiditis disseminata, strumous ophthalmia, ichthyosis, xeroderma pigmentosum, dermatitis herpetiformis, some variety of idiocy and imbecility, liability from neurotic causes to sudden rise of temperature, great susceptibility to attacks of all micro-organisms and zymotic poisons, night febrile delirium of children with temperatures of 99° to 101°, tubercular meningitis, Friedreich's disease, hydrocephalus, deaf-dumbness, &c. In the third period (7 to 13) of co-ordination of motion and emotion, one sees chorea, epilepsy, asthma, somnambulism, megrim, myopia, convergent strabismus, &c. Finally, during the fourth period, puberty and adolescence, the neuroses are: epilepsy, asthma, chlorosis, menstrual defects, hysteria, interstitial keratitis, megrim, adolescent insanity and stupidity, impulsiveness, dipsomania, perversion of the moral sense and volition, frothy religiosism, perverted sexual

instincts, unfounded aversion to relatives, arrested body-growth, adolescent ugliness, some kinds of joint diseases, ingrowing nails, acne, subunguial exostosis, naso-pharyngeal polypi, beardlessness, barrenness, phthisis, acute rheumatism, &c.

This simple enumeration shows what a number of questions are raised in this volume. One cannot feel uneasy, however, at the number and variety of complaints attributed to one and the same cause; and one recollects the teaching—not so very long ago—that typhoid fever was a developmental disease which young people had to go through. The author somewhat neglects, among the complex causes at work in the causation of disease, the external influences. A disease is always the result of two factors—of a certain innate predisposition and of an occasional cause. In some cases, at least, the second cause is of more moment than the first. It is thus for moral as well as physical phenomena. Evidently, a hereditary predisposition is necessary for the development of obssessions or hysteria. But it is nevertheless true, that great emotions, suffering, or afflictions have determined the disease and its peculiar form. The same patients placed under different circumstances would probably not have been affected in the particular manner they have. Inversely, other persons whom we consider to be healthy, would have become hysterical or otherwise affected if placed in the position of the former. We have to deal with a very complex mixture of physical and moral influences, of acquired educational transformations, and of original predispositions; and every factor must be equally held into account in our explanations of the phenomena.

The author no doubt is perfectly conscious of all this, but has been only led to exaggerate somewhat one factor, in his anxiety to demonstrate its importance. The book is not intended to account fully for the neuroses, but to study them from a special point of view. Clouston has thus been enabled to give some very curious cases, and viewed from a highly interesting aspect—chorea, megrim, epilepsy, hysteria (justly considered as a mental disease), and adolescent insanity. He has proved the capital importance of neuropathic heredity, and the consequences which flow from this notion with reference to the interpretation and treatment of these neuroses.

P. Janet, M.D., Paris.

L'Idiotie, hérédité et dégénérescence mentale psychologie et éducation de l'Idiot. Le Dr. JULES VOISIN, Médecin de la Salpêtrière, Paris, Alcan, 1893.

THIS book consists of a number of lectures on Idiocy delivered by the author at the Salpêtrière during the first quarter of the year, 1893, and is an excellent summary of the conditions known to exist in the disease. The first lecture deals with the subject of heredity in general, and more especially with that form of morbid heredity which under the name of mental degeneration has been so well described by Morel. Dr. Voisin shows that though all causes which weaken the system, such as intemperance in the use of alcohol, tobacco, and morphia, malaria, defective nourishment, vitiated air, poverty and debauchery, produce degeneration in the human species, yet this human degeneration assumes a particular character in the heredity of nervous and mental maladies, so that the most diverse affections appear, and succeed one another in a family during a long series of generations. Mental and nervous diseases which are essentially hereditary in character occupy the principal place among causes of human degeneration, but they are transformed in passing from parents to children; the degeneration increases in a more or less rapid manner to end in the last stage of idiocy and cretinism, which produce sterility. Consanguinity is often a principal factor in producing this degeneration, and nothing shows this more clearly than the history of the Royal Family of Spain, from 1449 to 1700, in which the bastards alone of these families possessed their faculties in a sane condition. After dealing with the congenital and acquired causes, the author proceeds to discuss the pathological anatomy of idiocy. The lesions met with may be accidental or evolutional in character, and are seen in the cranio-facial osseous system, and in the cerebro-spinal apparatus. Amongst the accidental lesions are injuries to the cranium, cerebral compression, hæmorrhages in the meninges or cerebral substance, embolisms and thromboses. The various shapes of the cranium, such as the microcephalic, scaphocephalic, plagiocephalic, dolicocephalic, &c., are mentioned, and reference is made to craniectomy as practised by Mr. Lannelongue, in cases of microcephalic idiocy. The author agrees with Mr. Bourneville that the operation is of little use, as the microcephalic head is a sign of a vice of nutrition which affects the trophic functions of

the nervous system. Facial asymmetry is often seen, as well as an arched condition of the palate, bad dentition, and badly-formed ears. The evolutional lesions of the cerebro-spinal axis are very numerous, and most important; they affect the whole or part of the brain, and are very variable in character. Hypertrophy of the brain is rare, but atrophy, sclerosis of parts of the substance, and hydrocephalus, are more commonly met with. True and false porencephalus are often seen in paralysed cases, the former being due to arrest of development, the latter the result of a destructive process due to a trouble of circulation supervening during intra-uterine life or coming on later. The author gives numerous definitions of idiocy, and concludes with one of his own:—"The idiot is an individual whose intellectual, sensory, and motor faculties are not developed, or are abnormally developed in a defective manner, or are arrested in their evolution before or some years after birth to a degree which they cannot overcome in consequence of various fœtal or chronic lesions of the brain." This, like other definitions however, does not give any idea of the part which etiology and pathological anatomy play in this affection; neither does it give an account of all the degrees of idiocy which one observes in practice. Various classifications are noticed, including that of the author, who divides idiocy into four categories, viz.: (1) complete, absolute idiocy, congenital or acquired; (2) incomplete idiocy, congenital or acquired, susceptible of amelioration; (3) congenital or acquired imbecility; and (4) mental debility. The symptoms and clinical aspect of idiocy are fully described, reference being made not only to the malformations of the cranium, asymmetry of the face, vaulted palate, and condition of the teeth and ears, but also to the condition of the limbs. The arms are often too long for the body, a condition similar to that found in monkeys, and the fingers may be webbed or increased in number; in addition, there is often paralysis with atrophy and athetosis. A long description is given of the conditions found in the genital organs, and we learn that puberty is not retarded in girls, but is delayed in boys. The author then proceeds to study the condition of the senses in idiots, and passes in review successively, sight, hearing, smell, taste, general sensibility and the muscular sense. With respect to sight, visual perception of objects is developed very late in infant idiots, and in some cases it is never present; the look is then always vague, and those affected in this way cannot by the sense of sight acquire exact notions on the form, relief and dimensions of

objects. Tamburini, Morselli and Benedikt have observed in these cases occipital flattening coinciding almost always with these phenomena; and as the organ of vision is generally sound, and these patients do not see, or see badly; the reason is that their visual centre is insufficiently developed. This centre, according to Munk and Monakow, has its seat in the occipital lobes, and this fact explains to us the co-existence of occipital flattening and psychical blindness. Reference is made to the anatomical description of the brain and cranium given by Lavocat and Franck, who consider the cranium as a continuation of the vertebral column, its osseous pieces representing modified vertebræ, which contain four distinct segments of nervous substance, the seat of the special senses; the occipital vertebra enclosing the sense of sight, the sphenoidal that of taste and smell, the temporal that of hearing and the frontal the seat of intelligence. The senses of taste and smell remain generally in a rudimentary condition; some idiots are gluttonous, others, with a perverted taste, eat anything which comes in their way, such as paper, earth and pebbles; in these the sense is very little developed and they cannot distinguish sweet from bitter substances. A blunted or perverted sense of smell is frequent, and patients will be seen to inhale with indifference the vapour of ammonia and of eau de Cologne; it is necessary, therefore, to keep out of their way all injurious substances. As to the sense of hearing, deafness is much rarer than blindness in idiots, and when present is due either to lesion of the central organ, which is the more frequent cause, or to defect in the auditory apparatus; on the other hand, many have the musical sense very well developed. The muscular sense is deficient, and associated movements, such as walking, taking hold of an object, and dressing are with difficulty performed. Some idiots are constantly moving about, others on the other hand are quite inert, and will remain all day in their chair or bed; others again are subject to various automatic movements, and others destroy everything that comes in their way. Tactile sensation, general sensation, and organic sensations are sometimes diminished and sometimes absent. The chapter on language in idiots is a very interesting one. Language is shown to consist of a concourse of intellectual, sensorial, and motor images associated in a definite fashion. When one pronounces the word "orange," for example, an auditory image is called up, which, in its turn, raises the visual image of a round object; this visual image creates an olfactory and gustatory image, and from the whole of these images results the idea that we

have of the word "orange;" if we wish to write or pronounce the word all the preceding images call up the special image of articulation for writing or for speech. Suppose, however, that one or many of these images are not awakened; in this case we shall have an incomplete, imperfect, or no idea of an object, and this is what happens in the case of idiots. The intellectual operations, which succeed one another at the moment of an auditory impression, are reviewed and arranged in three orders, according as the auditory perception is effected in a rude fashion, is differentiated, or becomes verbal. When these three forms of hearing are attacked separately, we have cortical deafness, psychical deafness, and verbal deafness. In idiots, who cannot speak, we have to distinguish aphasic motor idiots, from idiots who are aphasic from psychical deafness or verbal deafness. In the first case, the idiot cannot speak, but he comprehends what is said to him; in the second case, that of the profound idiot, he cannot comprehend or utter a single word; in the third, he cannot comprehend words, but can comprehend gestures.

Passing over the chapters on instincts, sentiments, intelligence, and will, we come to one on "idiotie myxœdémateuse" or "cachexie pachydermique," the names given by French writers to the disease which is known as sporadic cretinsim in this country. The well-known characters of this affection are described, and the author points out that it comprehends three orders of symptoms: (1) the cutaneous alterations which have given to it the name of pachydermic cachexia; (2) the arrest of intellectual development, the idiocy; and (3) the absence of the thyroid body. Even in those cases where the thyroid has been found to be hypertrophied, histological examination of it has shown that the hypertrophy of the connective tissue had caused atrophy of the proper elements of the gland, so that its function was suppressed. Reference is made to Mordret's researches, who found that in from 11 to 50 per cent. there was atrophy of the thyroid gland in idiocy, and who was of opinion that there was an intimate relation between thyroid atrophy and the degree of intellectual dulness. The author tries to explain the way in which the suppression or absence of the thyroid gland produces the disease, and on this point the experiments of Schiff and the observations of Ragowitch, of Albertoni, and Tizzoni are quoted. Dr. Voisin refers to the treatment of grafting portions of the gland, and injecting an organic liquid prepared from an extract of it, but makes no mention of the treatment now practised of administering by the mouth tabloids containing an extract of the gland.

The last chapter deals with the treatment of idiocy, and a full description is given of the physical, medical, moral, and educational treatment, now pursued in all institutions for the care and training of idiots and imbeciles. Great stress is laid on the prophylactic treatment, both as regards the parents and the patient, and the evils to be avoided by the parents and the means required for putting the patient into a healthy condition are fully pointed out. The book is well illustrated by cases which have come under the author's care.

FLETCHER BEACH.

Savill on the Electrical Changes of so-called Idiopathic Myopathy.

In the *Nouvelle Iconographie de la Salpêtrière*,[1] Dr. T. D. Savill publishes full particulars of an interesting case which corresponds in its history and clinical features with the Infantile Idiopathic Amyotrophy of Duchenne, but which, nevertheless, presents certain interesting changes in the electrical reactions of the muscles both to faradism and galvanism. Examples of this disease have generally been described as unattended by any electrical changes, and have consequently been regarded as due to inherent alteration in the muscle itself (a primitive myopathy), independent of any neuro-spinal lesion. The observation before us goes to show that this is not invariably so.

Cases of this class tend to gravitate, after visiting the different hospitals, to the metropolitan infirmaries, and Dr. Savill expresses the belief, founded on his experience at the Paddington Infirmary, that cases of so-called idiopathic or primary myopathy nearly always, at some time in their history, present electrical changes of some kind. This is contrary to the hitherto accepted view that this feature of unaltered electrical reactions is (after the age at which the symptoms first appear) the chief one which distinguishes cases of idiopathic myopathy from the larger group of progressive amyotrophic paralyses (anterior polio-myelitis). Dr. Savill further holds the view that many of the phenomena attending those cases are explicable on the assumption that there is a chronic generalised neuritis of very insidious onset and slow march. Certainly the case he

[1] May, 1894.

describes presented, at one time, tenderness and pain along the course of the nerves and electrical changes, which would tally with this opinion. A case which tends to support this view has has also been recorded by Guinon in the *Nouvelle Iconographie*,[1] and the number of the journal which contains the one under consideration mentions others. Brissaud also publishes several cases having the same bearing,[2] and maintains that the clinical distinction hitherto drawn between Primitive Muscular Dystrophies and the Amyotrophies of spinal origin are not borne out by the facts. Dr. Savill also points out that cases of "primitive myopathy" and of amyotrophy accompanied by the usual electrical changes occur in different members of the same family;[3] that cases of pseudo-hypertrophic paralysis, which undoubtedly belong to the class of so-called myopathies, are sometimes attended by marked electrical changes;[4] and that these circumstances all tend to support the idea that the lesion of the myopathies is, like the amyotrophies of spinal origin, primarily in the nervous system; though on account of its slight and, may be, evanescent character, it has hitherto escaped detection.

The patient was a girl named Susan S., admitted, on November 6, 1889, into the Paddington Infirmary, where she remained under observation for five years, and where she still drags on a weary existence.

The family history on the mother's side was healthy, but on the father's there was a history of epileptic and other fits, paralysis, and a tendency to drunkenness. An elder sister of the patient had certainly, from the clear account given by the mother, been attacked with identically the same form of amyotrophy, and another sister had shown some suspicious symptoms of it.

The patient, according to the mother's account, had never, even as a baby, been able to shut her eyes properly, but as a child she was bright and intelligent, and nothing abnormal was noticed about the muscles.

At the age of 7 she showed weakness and curving forwards of the spine, for which she was treated by lying upon the back, and about the same time it was noticed that the face was becoming

[1] January, 1893—a case under the care of M. Brissaud in 1890.

[2] "Leçons sur les Maladiè Nerveuses," Paris, 1895, p. 344, &c.

[3] See also Charcot, "Lectures on Diseases of Nervous System," New. Syd. Soc., vol. iii., p. 64, and Cenas and Douillet, *Loire Médicale*, Nos. 7 and 8, 1885.

[4] Bédard et Rémond, *Arch. Gen. de Med.*, July, 1891.

expressionless, the speech altered, and that the lower lip was becoming very protuberant.

When about 8 years of age the mother was attracted by the commencing deformity of the feet. At the age of 9 the deformity and weakness of the legs was sufficient to prevent her from walking, and an operation was suggested but refused. She was never able to walk after this.

At the age of 10, though the feet were deformed, there was no general wasting obvious to the mother, and the patient was a fair height for her age. However, the weakness of the legs and face was becoming progressively worse. The involvement of the arms was so gradual that it is unknown when the deformity and wasting of these first began. It seems probable, therefore, that the atrophy started in the face as a baby, and, about the age of 7 or 8, attacked the back and legs.

Condition on Admission to the Paddington Infirmary in November, 1889, *aged* 18.—The first of the pictures shows the atrophy of arms and legs, the dropped hands, and the talipes equinus.

Figs. 2 and 3 show the same deformities, and the marked spinal lordosis in other attitudes. Fig. 2 shows very distinctly in the profile the protruding lips, which the patient was quite incapable of closing. The difficulty of supporting her accounts for the blurring of the outline of fig. 3. They need no further description. At this time there was some hyperæsthesia of the skin and some general tenderness of muscles and of the distal segments of nerve trunks.

Fig. 4 shows the maximum amount of closure of the labial and visual orifices. There was at this time an extreme degree of amyotrophy, which is described in considerable detail (*loc. cit.*).

One of the most noticeable features of this case, like others of the same kind, was the marked predominance of the atrophy of the extensors over the atrophy of the flexors, resulting in "dropped hand" and "dropped foot." These deformities were due to the tonic contraction of the remaining healthy fibres of the flexor muscles, for they could be straightened almost completely by passive movement. The power of flexing the extremities was retained to some extent, but the power of extension was almost *nil*. The extremities were always very cold and livid.

Electrical examinations, both by Dr. Savill and Dr. Kilner, showed a general *diminution of faradic contractility*. Most of the muscles required three, four, or even five times the normal strength

of current to produce contraction. A notable exception to this was found in the flexors of the forearm and the flexors of the lower leg on both sides of the body, which give a reaction with about a fourth of the normal strength of current, showing thus an increased faradic irritability.

The *galvanic contractility* was also, in general, somewhat diminished, the muscles requiring two or three times the normal strength of current to produce effect. Here again a notable exception was found in the flexors of the forearm on both sides, and the flexors of the left lower leg, where the contractility was somewhat increased, and KCC approximately equalled ACC. The galvanic contractility of the flexors of the right lower leg were normal, both quantitatively and qualitatively. The galvanic contractility was also, in general, *qualitatively* altered so that KCC and ACC were too nearly alike, the one being barely double the other instead of $3\frac{1}{2}$ times, the only exception being the flexors of the right lower leg, as just mentioned.

Aged 19.—When the patient was admitted, at the age of 18, there were some general tenderness of the muscles and nerve trunks, and some hyperæsthesia of the skin; but about the age of 19 she developed, in addition, attacks of severe pain, accompanied by acute tenderness along the nerve trunks. These symptoms became less the following year, but never entirely disappeared. Fibrillar tremors, such as Duchenne describes in these cases, were not observed in this patient, but the limbs were affected with constant fidgety movements, and an inability to keep in any one position, resembling athetosis — another symptom which might be regarded as pointing to an involvement of the nervous system.

Aged 23.—But little change had taken place in the patient during the five years she had been under observation. She kept to her bed, the deformities seemed to be gradually getting more pronounced, and the weakness greater. The muscles were again examined by Drs. Kilner and Savill. There was, as before, a marked general diminution of *faradic contractility*, with the exception of the flexors of the forearm on both sides, where there was a slight increase, and excepting also the flexor muscles of the left lower leg, where the faradic contractility was approximately normal.

As regards *galvanic contractility*, there was a greater general

FIG. 1.

FIG. 2.

Fig. 4.

Fig. 3.

diminution than on the previous occasion, with the same exceptions as before, namely, the flexors of both forearms, and now *both* lower legs, where the contractility was fairly normal as to quantity; but KCC and ACC were now equal. Over the rest of the body KCC was only a little greater than ACC. These results are embodied in two tables which accompany the paper.

In general terms the degree of alteration of the electrical reactions was proportionate to the loss of muscular substance and power. In those muscles, however, which retained some degree of power, there was, in addition to a diminution to both currents, a qualitative alteration of the galvanic contractility. On the occasion of the second examination, the muscles which retained most power were the flexors of the legs and arms. With these exceptions the degree of atrophy was extreme.

A copious bibliography on the subject of amyotrophy is given at the end of Dr. Savill's paper. A classification of the amyotrophies is also given.

Mott and Sherrington on the Influence of Sensory Nerves upon Movement. (Abridged from the *Proceedings of the Royal Society*, 1895, p. 481.)

(1) *Effect of Section of the whole Series of Sensory Roots belonging to the Limb.*—By the "whole series" is meant in the brachial region from the fourth cervical to the fourth thoracic inclusive; in the lumbar from the second to the tenth post-thoracic inclusive.

From the time of performance of the section onwards, as long as the animal may be kept, the movements of the hand and foot are practically abolished; the movement of grasping, which is so frequent and useful to the monkey, both with the hand and foot, never occurs at all in our experience. On the other hand, the movements at the elbow and knee, and especially the movements at the shoulder and hip, are much less impaired. The fore limb hangs from the shoulder partially flexed at the elbow; the hind limb is flexed at hip and knee. As the animal runs about it does not attempt to use the leg; the fore limb swings helplessly, with flexion at elbow and wrist and adduction at shoulder, in much the same position as if carried in a sling. The hind limb looks as if it were being held up so as to be kept off the ground while the

animal runs on three legs; we are inclined to think that this appearance is deceptive, and that the position results from an equilibrium of the action of the muscles, in which purposive action on the part of the animal does not play a *rôle*. When the animal is allowed to climb a rope or the side of the cage, the fore limb swings more or less helplessly, and is not used for the climbing; similarly, the hind limb is kept more or less flexed at hip or knee, and is not used for the climbing. If the feeding-time be deferred and an animal, in which the *apæsthete* limb is an arm, be tested by offering it fruit after the sound arm has been secured behind the back, there is no attempt to use the apæsthete limb for reaching the food, but the neck is thrust forward in order for the mouth to seize it. If the fruit be placed in the hand of the apæsthete arm, the animal does not lift the hand, and appears quite unable to do so, even though encouraged. If, however, the hands of a tame normal monkey be secured behind its back, and as it lies on the floor, fruit be placed near it, the fruit is usually taken at once with the foot; but if the leg is apæsthete the fruit cannot be taken, although in one monkey the attempt used to be made. The foot was rapidly thrust toward the fruit by extension of hip and knee, but the foot missed its object widely, *i.e.*, by several inches, and the digits were not moved, though the ankle appeared to be slightly plantar-flexed. The impairment of motility in the limb ensues immediately upon completion of the section; that is to say, directly the effects of the anæsthetic have passed off sufficiently to allow requisite examination of the animal's ability to move its limbs, the above-described inability is discoverable as fully developed as at any subsequent period. We have kept the animals alive for various periods up to and over three months, and there has been no obvious change in the condition, either in the direction of improvement or the reverse. In the case of the lower limb, after two or three months, the constant position of flexion of hip and knee, on two occasions, gradually induced a change in the muscles of the thigh, which prevented hip and knee being properly extended, even by passive stretching.

As to the nature of the disturbance of motility in the limbs, one feature, namely, its peculiar topographical distribution, is salient and constant. The defect in motility increases from the attached base to the free apex of the limb; so that, for instance, while comparatively slight at the hip, it is successively greater at knee and ankle, and greatest (amounting as regards volition to absolute loss) in the digits.

In this respect it curiously closely simulates the impairment of motility ensuing upon ablation of the limb region of the cortex cerebri; but it is, in the monkey, somewhat *more severe* than the impairment following cortical ablation.

We find, however, that forcible and rapid movements, even of the fine joints at the end of the limb, can be induced in the animals by causing them to "struggle"; for instance, while recovering from ether inhalation, or while trying to free themselves on being held awkwardly, the whole limb at all its joints may exhibit movements; but even under these circumstances it is only once or twice that we have seen "grasping" movements of the digits, although sharp extension of the digits is not nearly so infrequent.

We are led from these and other considerations, which will be detailed in a fuller paper, to conclude that *associated movements in the limb* ("Mitbewegungen") *are comparatively little impaired* by loss of the sensation from the limb in which they occur; but that the independent and more delicately-adjusted movements which employ preponderantly the smaller and more individualised muscular masses of the hand and foot, and serve to move the digits, especially the hallux and the thumb—in fact, just *those movements which are represented most liberally in the limb area of the cortex, are extremely severely impaired, and, in some instances, are abolished.* We say "abolished" advisedly, because we are persuaded by our observations that, in the case of certain movements, *e.g.*, grasping movements of the hand and foot, opposition of pollex and hallux, the animal is rendered absolutely powerless to perform them, even under the strongest possible inducements. This conclusion has been gradually forced upon us. Although we are aware of the danger of introducing terms relating to consciousness into descriptions based almost solely on motor reactions, we believe that we cannot more lucidly state the condition of the animals than by saying that the volitional power for grasping with the hand, &c., had been absolutely abolished by the local loss of all forms of sensibility experimentally produced. Further, that this volitional power was lost immediately from the time of operation, and that there was not the slightest evidence of any recovery of it during the longest periods to which our observations extended (about four months).

This being so, it is natural to inquire what influence, if any, is exerted by the section of the posterior spinal nerve-roots of the limbs upon the reactions obtainable from the limb area of the cortex? That no diminution, but rather a slight increase of the excitability of the cortex, is the immediate result has been shown

by one of us previously.[1] But the question remained, what will be the result when, for many weeks, the severance of the roots has led (as above shown) to disappearance from the limb of those very movements which the cortex, when experimentally excited, is especially able to produce? We have answered this, both by electrically exciting the cortex and by giving absinthe intravenously to produce epilepsy (Magnan). On exciting the cortex cerebri of the hemispheres in the appropriate regions for eliciting movements of the thumb, hallux, or digits, the responsive movements have been as easily elicited from the apæsthete limb as from the normal limb, and it has several times seemed to us rather more easily, that is to say, with a slightly less intensity of faradic current (the rate of interruption always remaining the same, $\frac{1}{30}''$).

As to the absinthe epilepsy, it always affected the apæsthete limb in a manner not distinguishably different from the normal limb. Convulsions sometimes started in the normal and desensitised limb simultaneously, sometimes a little earlier in one or the other; but no indubitable predominance or preference was shown by either limb. In a very few of our experiments (three) no movement was obtained in the apæsthete limb on excitation of the cortex; this was found to be explained subsequently by naked-eye degeneration of the pyramidal tract as revealed after hardening in Müller's fluid. This degeneration was due to injury accidentally inflicted upon the lateral column of the cord by the operation. The spinal tonus in the muscles of the apæsthete limb is undoubtedly much diminished.

These observations seem to us to point to the profound difference existing between the production of the finer movements of the limb in volition on the one hand, and by experimental stimulation of the cortex on the other. The fundamental importance of sensation for those finer movements of the limb, which are so especially well represented in the cortex of the ape, has by no authority been more forcibly emphasised than by Dr. Bastian. We think these experiments go even further than his arguments in pointing to the influence of sensation upon voluntary movement, inasmuch as they indicate that not only the cortex, but the whole sensory path from periphery to *cortex cerebri*, is in action during voluntary movement.

(2) *Effect of Section of a Single Sensory Root.*—In striking contradiction to the above-stated impairment of movement in the

[1] C. S. Sherrington, *Phil. Trans.*, vol. clxxxiv., B, pp. 690, 691.

limb ensuing upon section of the whole series of its sensory nerve roots stands the effect of section of any one of the sensory nerve roots of the series singly and alone. In the latter case no impairment of movement at all results, or, at least, can with certainty be detected.

This is the case even when the largest and most important sensory root of the series is chosen for section; namely, in the upper limb the eighth cervical, and in the lower limb the sixth post-thoracic. (These are the nerves that supply the skin over the whole of the hand and foot respectively. It is to be remembered, however, that hand and foot respectively are each of them supplied with sensation by at least three sensory roots, the middle root covering the whole surface in each case.)

We attribute the fact that section of these large roots with their wide distribution over hand and foot produces so little appreciable effect, to the fact that the distribution of all the spinal nerves in the skin is an overlapping one. The extent of overlapping is great enough to prevent the section of any one nerve, even of the largest, producing actual anæsthesia of the skin in any part.

We further find that even if a field of absolute anæsthesia be actually produced by section for instance, of the seventh, eighth, and ninth post-thoracic roots, or, in some cases, by section of the seventh and eighth cervical and first and second thoracic roots, the impairment of movement resulting in the limb is comparatively slight. This is the more remarkable when the region deprived of sensibility includes some of the most highly sensitive parts in the limb, namely, those of the palm.

In such a case the retention of sensibility, although in an impaired degree, by the radial side of the palm, including pollex, is the cause, in our opinion, of the remarkable quantity and quality of movements still executed by the limb in spite of that impairment. In the case of section of the seventh, eighth, and ninth post-thoracic sensory roots, it was almost impossible, even on the closest examination, to detect any defect of movement whatever; the animal used both its feet apparently equally well in climbing or running, there was no clumsiness in picking up small objects (as a kernel) with the foot, and no hesitation in so doing. The chief detectable muscular difference between the limbs was that the knee-jerk was more brisk upon the operated side, and the calf and ham and back of the thigh were very deficient in sensation.

In the case of section of the sensory roots of the seventh and

eighth cervical and first and second thoracic, besides the ulnar border of the arm, the hand, with the exception of the thumb and radial side of index, is also quite insensitive; and the sensitiveness of the skin of the thumb and index, where present, is below normal in degree. Nevertheless, although impairment of movement (especially clumsiness) is obvious, yet the condition is strikingly different to that obtaining after the whole series of roots has been cut through. Food is easily taken up from the hand and picked up from the floor with the thumb and index of the partially apæsthete limb, and the limb is freely used in progression and in climbing. The grasp of the partially apæsthete hand is easier, however, to detach from the cage than that of the normal hand.

We find, again, that a degree of impairment very nearly as great, indeed not obviously different from that produced by section of the sensory roots of the whole series of the nerves of the limb, is produced by section of just those sensory roots which supply the apex of the limb, *i.e.*, the hand and foot. If the whole hand or foot be completely apæsthete by the sections, then the peculiar degree of inability to move the limb, described at the outset of this communication, is obtained in (as far as we can see) its full extent. This result has its converse in the following, which we have also noted:

(3) *Effect of Section of the whole Series of Sensory Roots belonging to a Limb, with the exception of a Root that supplies the Hand or Foot.*—If the sensory roots of the whole series of the spinal nerves belonging to a limb be severed, with the single exception of, in the upper limb, the eighth cervical (distributed to the whole extent of the hand), and in the lower limb the sixth post-thoracic (distributed to the whole extent of the foot), a certain degree of impairment of movement of the limb results, which appears rather as weakness than clumsiness, but the degree of impairment is altogether quite slight. The limb is used freely for progression, for climbing, for picking up food, and bringing it to the mouth. The grasp of the partially apæsthete hand is easier to detach from the cage than that of the hand of the normal side.

The question naturally arises whether in the interruption of the paths of afferent impulses these can be so dissociated as to decide what share the muscular sense takes in the results observed? The afferent nerve fibres from muscles *per se* run, in every case in which they have been examined, in the corresponding spinal roots to the motor. It is possible, therefore, in the hand and foot to sever the sensory roots supplying the muscles

while only partially interfering with those supplying other structures—the skin, sheaths of tendons, joints, &c. The converse is also true. On leaving intact the afferent fibres from muscle for the greater part, but interrupting all other afferent channels, we find that the defect of movement produced is nevertheless extreme, as, for instance, when the lowest four cervical and the first thoracic roots are severed. In the same way, when the fifth, sixth, and seventh post-thoracic roots are divided, the sole of the foot is apæsthete, and the defect in movement is extreme, although the afferent fibres from the plantar muscles remain to a large extent intact.

Conversely, when the seventh, eighth, and ninth post-thoracic roots are severed, the afferent fibres from the plantar muscles are completely interrupted, but the sole of the foot remains sensitive all over, owing to the sixth and fifth roots. In this case (and we have two instances of the kind), the defect of movement, if it exists at all, is not appreciable.

It must be remembered that other deeper structures are innervated by the same nerves as the skin, and these we do not wish to dissociate from the sensory channels still uninjured after interruption of all afferent tracts from the muscle itself.

One of our chief results is:—That afferent impulses, both from the skin and from the muscles, especially the former, as related to the palm and sole, are necessary for the carrying out of "highest level" movements. From observations on tame animals we are firmly convinced that there exists actual inability (and not only absence of volition) to perform the movements in question.[1]

Bastian on the Relations of Sensory Impressions and Sensory Centres to Voluntary Movements.

In a subsequent part of the *Transactions* Bastian criticises Mott and Sherrington's conclusions. After giving a *résumé* of his views on Kinæsthesis, he says:—"From the point of view of sensory guidance, cortically initiated movements are divisible into two categories:—(1) *Speech movements* which are now known to be produced under the guidance of co-active auditory and kinæsthetic centres; and (2) *Limb and other bodily movements*, which are equally well known to be produced under the guidance of co-active visual and kinæsthetic centres. In each case the kinæs-

[1] *Cf.* Chauveau's article on "The Sensorimotor Nerve-circuit of Muscles," BRAIN, 1891, p. 145.—ED.

thetic centres do not seem to act independently, but only in response to the primarily initiated activity in the auditory or in the visual centres respectively—these latter in all cases seeming to take the lead."

Limb movements may be paralysed by organic lesions or by functional defects. The latter, again, may be either cerebral or spinal in seat. Bastian has described cerebral functional paralyses in the volume, of which a review is given in the present part of BRAIN. Of the *spinal functional defects* (also discussed in that part of review) he speaks as follows :—

"It is into this catagory that, in my opinion, the forms of paralysis fall that have been produced by Drs. Mott and Sherrington as a result of section of all the sensory roots of a limb.

"We have been long familiar with forms of paralysis due to structural lesions affecting the great ganglion cells in the anterior cornua of the cord; those, for instance, due to polio-myelitis or to focal lesions of different kinds involving the anterior cornua.

"Three years ago, moreover, I gave reasons for believing that there are cases of functional paralysis of spinal type, due to defects in these same regions of the spinal cord (examples of which were recorded), that ought to be distinctly separated from those of cerebral origin commonly known as "hysterical." We have now, as I am inclined to maintain, in the experiments of Drs. Mott and Sherrington, an experimental proof of the existence of one of these forms of functional paralysis of spinal origin.

"Instead of a lowered functional activity in cerebral kinæsthetic centres (interfering with volition), we have here a lowered functional activity in the motor centres themselves situated in the spinal cord, by reason of which their molecular activity is so altered that they are no longer capable of responding to ordinary volitional stimuli coming from the cerebral cortex. This, at least, is the explanation which I would suggest, instead of supposing, as the authors do, that volitional power itself 'has been absolutely abolished by the local loss of all forms of sensibility' in the paralysed limbs.

"As I have already stated, a local loss of all forms of sensibility caused by a lesion in the brain in the region of the internal capsule causes no such paralysis. There is, therefore, no ground for supposing that the animal's power of willing is interfered with by section of all the posterior roots coming from the limbs; while, on the other hand, all the details furnished by Drs. Mott and Sherrington are quite in harmony with the interpretation that the animal's ordinary will-power is unable to excite the spinal motor

centres to action when their molecular condition has been altered by cutting off all the different stimuli proceeding to the corresponding region of the cord. The immediate result of this may be presumed to be a lowering of the habitual sub-activity of the motor centres upon which the condition of *tonus* in the muscles depends.

"The fact that the results are produced only when *all* the sensory roots are cut, and that 'the defect in motility increases from the attached base to the free apex of the limb,' so that 'the independent and more delicately adjusted movements which employ preponderantly the smaller and more individualised muscular masses of the hand and foot' are those which are most severely affected or abolished, although they may appear confirmatory of the interpretation of Drs. Mott and Sherrington, are, in fact, no less in accordance with that here given. This will be seen to be so if we bear in mind the overlapping of the fields of distribution of the sensory roots in the spinal cord (as shown by Sherrington), and the fact that the most delicate stimuli going to the smaller muscles might be expected to be those which would prove most impotent to rouse the sluggish spinal centres into activity. Again, when the authors say: 'We find, however, that forcible and rapid movements, even of the fine joints at the end of the limb, can be induced in the animals by causing them to "struggle"; for instance, while recovering from ether inhalation or while trying to free themselves on being held awkwardly, the whole limb at all its joints may exhibit movements,' we have facts quite compatible with my interpretation. Muscles may not respond to ordinary volitional stimuli, and yet may respond when the stimulus is strengthened under the influence of emotion. In a similar manner may we explain the fact that when the kinæsthetic centres corresponding with an apæsthete limb are stimulated by electricity, movements of the previously paralysed limb are produced just as easily as they are in a normal limb.[1] Such results may, as the authors say, be considered to point to

[1] This fact shows that the cutting off of the afferent impressions by section of the sensory roots does not entail a lowered excitability of the kinæsthetic centres in the cortex, but rather the reverse, if we look to the really lowered activity in the spinal centres which the absence of tonus implies. In the case of complete cerebral hemianæsthesia, however, there is, over and above the absence of any such lowered activity of the spinal centres, another important difference between the conditions existing and those which obtain in the experiments of Drs. Mott and Sherrington, viz., no cutting off of cerebellar influence, so that the activity of the cortex and of the sub-cortical centres is less interfered with. These differences tend, I think, to explain the presence of paralysis with section of the posterior roots, and its absence with cerebral hemianæsthesia. (May 4, 1895.)

the 'profound difference between the production of the fine movements of the limb in volition, on the one hand, and by experimental stimulation of the cortex, on the other.' This explanation is as valid for the one as for the other interpretation.

"The stimulation by electricity may, and probably does, involve a very different kind of stimulus from that which normally emanates from the cortex during a voluntary act, and how subtle are the differences that exist between the conditions leading to paralysis or not in different instances may be judged from the functional cases of paralysis of cerebral origin, previously referred to, in which there may be in the same person, in immediately successive periods, complete paralysis of the limb so long as the eyes are closed, and no such paralysis when the eyes are opened."

J. B. Charcot on Progressive Muscular Atrophy. (Thesis, Paris, 1895.—Abst. in *Rev. Neur.*, Sept. 15, 1895.)

Several recent observers have contested the existence of this disease as an independent type. The author's object is to refute their arguments, by showing that its evolution and lesions are peculiar to itself; and that the lesions of the white fasciculi depend upon the poliomyelitic process itself. Five personal cases are described; two with *post-mortem* examination. The fifth is one of subacute anterior spinal paralysis, which the author connects with the other type.

In the two *post-mortems* he found the usual antero-cornual lesions, together with sclerosis of the anterior fundamental fasciculus. This is seen, on a transverse section, as a comma-shaped band, encircling the anterior horn, and tailing off backwards in the lateral column, parallel with the contour of the cord. This peculiar zone of degeneration has been casually noticed by previous observers.

The author shows its importance and explains it as due to the destruction of the fibres originating in the "cellules de cordons." Its localisation is similar to that of the supplementary degeneration of amyotrophic lateral sclerosis. It is found in other cases of poliomyelitis; though it may be slight, and escape discovery when Pal's method is used; carmine shows it well.

The author concludes that there exists a Duchenne-Aran type

of progressive muscular atrophy, as propounded by his father, the late Prof. Charcot. The sclerosis of the white fibres is no argument against it, since it depends upon the existence of a poliomyelitis, of which it forms a secondary degeneration.

Third International Congress of Psychology. (Munich, August 4-7, 1896.)

PROGRAMME OF WORK.

I. *Psychophysiology.*—(*a*) Anatomy and physiology of the brain and of the sense-organs (somatic basis of psychical life).

Development of nerve-centres; theory of localisation and of neurons, paths of association, and structure of the brain.

Psychical functions of the central parts; reflexes, automatism, innervation, specific energies.

(*b*) Psychophysics. Connection between physical and psychical processes; psychophysical methods; the law of Fechner. Physiology of the senses (muscular and cutaneous sensibility, audition, light-perception, audition colorée); psychical effects of certain agents (medicines)—Reaction-times—Measurement of vegetative reactions (inspiration, pulse, muscle-fatigue).

II. *Psychology of the normal individual.*—Scope, methods and resources of psychology—Observation and experiment—Psychology of sensations—Sensation and idea, memory and reproduction—Laws of association, fusion of ideas—Consciousness and unconsciousness, attention, habit, expectation, exercise—Perception of space (by sight, by touch, by the other senses); consciousness of depth-dimension, optical illusions—Perception of time.

Theory of knowledge—Imagination—Theory of feeling—Feeling and sensation—Sensual, æsthetical, ethical and logical feeling—Emotions—Laws of feeling—Theory of will—Feeling of willing and voluntary action—Expressive movements—Facts of ethics—Self-consciousness—Development of personality—Individual differences.

Hypnotism, theory of suggestion, normal sleep, dreams—Psychical automatism—Suggestion in relation to pædagogics and criminality; pædagogical psychology.

III. *Psychopathology.*—Heredity in psychopathology; statistics—Can acquired qualities be transferred by inheritance?—Psychical relations (somatic and psychic heredity), phenomena

of degeneration, psychopathic inferiority (insane temperament)—Genius and degeneration; moral and social importance of heredity.

Psychology in relation to criminality and jurisprudence.

Psychopathology of the sexual sensations.

Functional nerve-disease (hysteria and epilepsy).

Alternating consciousness; psychical infection; the pathological side of hypnotism; pathological states of sleep.

Psychotherapy and suggestive treatment.

Cognate phenomena: mental suggestion, telepathy, transposition of senses; international statistics of hallucinations.

Hallucinations and illusions; imperative ideas, aphasia and similar pathological phenomena.

IV. *Comparative Psychology.*—Moral statistics.

The psychical life of the child.

The psychical functions of animals.

Ethnographical and anthropological psychology.

Comparative psychology of languages; graphology.

[Further particulars and forms of application may be obtained from Professor Sully, East Heath Road, Hampstead.]